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Archives of Neurology and Psychiatry

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THE LANGUAGE OF SCHIZOPHRENIA

WILLIAM A. WHITE, M.D.

WASHINGTON, D. C.

It requires but a moment's serious consideration to realize that the subject of schizophrenic language must be immense if for no other reason than that it involves an understanding of the whole subject of language of which it is but a part. The extent and depth of the subject of language may be further appreciated from the fact that the single feature of its neuronic background as it is brought to attention in aphasia constitutes one of the most complex problems in the whole field of neurology and one with respect to which we are still hopelessly ignorant, especially when the enormous amount of work that has been done in this field is considered. In the face of these considerations it is evident that I cannot hope to do more than offer some few suggestions of perhaps minor import in themselves although they may be of more than passing significance as indicating the importance of the problems involved and to some extent, at least, the directions in which we may naturally expect to find material of value and something of the nature of the material that may be expected.

In the past the language of the schizophrenic, because of its frequently bizarre characteristics and its nonunderstandable and incoherent aspects from the point of view of the hearer, was supposed to correspond to thought processes that were similarly bizarre, meaningless and incoherent, and it has only been in recent years, since the comparative method has come to be applied in psychopathology and since the surface of thought has not been accepted as final for its structure, any more than the surface of the body has been considered as offering a full explanation of its anatomy, that we have begun to appreciate that what may appear to be without meaning may, as a matter of fact, be full of meaning if we are wise enough to find the key to its interpretation. The language of the psychoses and its interpretation is not without analogy to the inscriptions of unknown languages and their final translations.

Up to the present, however, we will find, if we search the literature, an amazing lack of serious consideration given to this obviously most important means of expression despite the development of philology and despite the generally recognized significance of language as a distinguishing human attribute.

In the first place, it would be necessary to a full treatment of the subject of the language of schizophrenia to preface that treatment by a consideration of language in general and this would inevitably lead to a full consideration of thought of which language is an expression and so to a quite complete treatment of the whole subject of psychology.

While this is manifestly impossible it does seem necessary to preface any consideration of the subject by a few brief statements regarding language and thought in general for unless we do we run the risk of taking over to our subject fragments, at least, of the outworn metaphysical and static concepts with which psychology is even yet sadly hampered.

In attempting to do this we are immediately met by certain tacit assumptions that belong to the category that I have designated, because of their very dangerous implications for our thinking, the pitfalls of the obvious. Among these tacit assumptions are these: that the world corresponds to our ways of thinking about it, and that language expresses these ways of thinking, and therefore the qualities as they exist in the world, quite accurately. I cannot undertake an exhaustive discussion of these points but must rest with urging the dynamic point of view as I see it.

To put it tersely, the idea that the world is as we see it and that language conveys from one to another an accurate account of things is one of those absolutistic concepts of which man has always been so fond because he is always wanting to settle things for once and all: he is always seeking rest in final solutions. But final solutions, like so many things that seem desirable, are but wishes for they nowhere exist and if they did they would not be desirable after all for they would spell death, not life.

For example, it seems obvious that there is no similarity between the redness of the rose that we perceive and a certain rapidity of vibrations in a hypothetic medium—the ether. One set of occurrences are physical phenomena, the others are mental, two separate categories which are incommensurable one in terms of the other. Similarly the world is full of individual men but there is no such thing as man in the abstract and yet language is replete with such abstract terms. To put these matters in a few words I will quote Hobson [1] who says: "It must, however, be observed that the experience of an individual is, in its fullness of detail, unique and incommunicable; it is only incompletely made intelligible to other individuals by means of language or other forms of symbolism. Thus language, in its very nature, involves abstraction in which the elements of actual individual experience are

1. Hobson, E. W.: The Domain of Natural Science, New York, the Macmillan Company, 1923.

replaced by symbols which fail to represent with absolute completeness what they are designed to describe."

It is manifestly impossible to discuss language quite separately and distinctly without considering, by implication at least, thought, of which language might be said to be the instrument. In the most primitive animals there is nothing that corresponds to thought. These animals are truly systems of reflexes. In them action follows immediately on the heels of stimulus. There is no hesitation, no intervening delay. The translation from stimulus to reaction is immediate, smooth and effective.

As we advance in the course of evolution to more complex, higher types this immediate translation of stimulus into action is ever more and more interrupted until, not only the interval between the two is or may be considerable but the resulting reaction becomes less and less predictable. It is in this interval between stimulus and action that thought occurs, that ideas and concepts are formed, and formulated into symbols.[2]

Presumably this occurrence of thought, this prolongation of the time between stimulus and action, this increase in indeterminateness of the action, are due to increasing complexity of the problems of adaptation and the introduction of more than one possible reaction to any given situation. As a result the several possibilities are weighed, there is doubt, examination of the advantages of the several possibilities, and decision, all of which corresponds, in physiologic terms, to the choice of the final common path.

In the primitive reflex organism the nervous pathway from receptor to effector is a single uninterrupted nerve fiber. The conditions that are going to make thought possible probably made their first appearance on the stage with the advent of the synaptic type of nervous system.[3] With the possibility of the nerve current being shunted in any one of several directions at the synapse the conditions were introduced that made for hesitation, increase in time interval, delay due to closely balanced forces that do not make for one path much more strongly than another, indetermination, in short for qualities which are associated with thought.

Now if, as we have seen, our thoughts and our symbols are not replicas in any sense of reality—what does take place in this region? The object of thought is action and it is only when the circumstances

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2. "Now it is, psychologists tell us, just in this interval, this space between perception and reaction, this momentary halt, that all our mental life, our images, our ideas, our consciousness, and assuredly our religion and our art, is built up." Harrison, J. E.: Ancient Art and Ritual, New York, Henry Holt & Co., 1913.

3. Herrick, C. J.: Neurological Foundations of Animal Behavior, New York, Henry Holt & Co., 1924.

are highly complex and the forces many and working in different directions that there occurs that delay that permits of thought. Thought and its instrument language do not portray reality; they are rather ingenious dexterities that enable us to find our way about.[4] So long as they serve this purpose they are useful and true; but to the extent that they fail, they lose both their usefulness and their truth. A study of language shows the constant discarding of symbols, the acquisition of new symbols and the changing meaning of symbols that continue to be used for a considerable period.[5] The responsiveness of the symbol to changing conditions, its capacity to take on new meanings under different circumstances, is of the utmost importance for our inquiry, for we find that schizophrenic thought, which is quite different from ours, makes use largely of the same words that we do in its expression.

After this somewhat overlong preliminary which I felt to be necessary in order adequately to background the subject let us proceed to the actual matter of the paper—the language of schizophrenia.

There is almost nothing in the literature on the subject although there is some literature on schizophrenic thought processes from which the language can, of course, to some extent be inferred. Take one of the more recent textbooks for example, that of Bleuler. His emphasis is on the peculiarities of association and he mentions in particular the weakening of the associative links so that the thought components tend to fall apart and so to lack coherence. He discusses also "crowding of thoughts," brevity of associations and perseveration. He particularly refers to the disregard of the time element in associations and recognizes such mechanisms as condensation, displacement, symbolism. His account is, on the whole, a mixture of description with here and there attempts at explanation such as the explanation of the neologism "gruesor" as a condensation derived from gruesome and sorrowful.

One of the early attempts to read meaning into schizophrenic expressions was that of Bertschinger.[6] In attempting to account for the recovery of schizophrenic patients he described three mechanisms he found in use: (1) correction of the delusions; (2) resymbolization, and (3) evasion of the complex. These mechanisms obviously imply each their appropriate language but I will stop with merely mentioning them as it is fairly obvious from their designation about what they mean.

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4. Vaihinger, H.: The Philosophy of "As If," New York, Harcourt, Brace & Co., 1925.

5. The value of the symbol in the course of development may be found discussed in White, W. A.: Mechanisms of Character Formation, New York, the Macmillan Company, 1920.

6. Bertschinger, H.: Processes of Recovery in Schizophrenics, Psychoanalyt. Rev. 3:176 (April) 1916.

There have been a few more recent attempts to interpret schizophrenic thought and language on which, because they are somewhat more directly aimed at the problem and because they take into consideration certain correlative data, I will dwell somewhat more at length.

The first article of importance is that of Schneider.[7] He discusses primarily the differences between the reproductive speech disturbances of aphasia and the domain of schizophrenic disturbances which are to be sharply distinguished. His effort is in the main descriptive. I cite it because of its interest from this standpoint and because it is one of the few serious attempts to study schizophrenic speech. He concludes: "In aphasia the speech disturbances are always in the form of imperfect construction; in schizophrenia when the train of thought is recognizable there is either no disturbance of speech or they take the form of ellipses, if the train of thought is not recognizable there are still neither literal or verbal paraphasias or amnesic-aphasic phenomena. In sensory aphasia there are gaps due to dropping out of words but with no noticeable regularity; in schizophrenia either entire members of the sentence are wanting or members of a certain type are preserved. In agrammatic sensory aphasia the ideas stand in proper relation to one another, the difficulty is one of expression. In schizophrenia remnants and isolated words are without connection (word salad). In aphasia there is no tendency to saltatory speech and no derailment due to flight of ideas. In schizophrenia there is no paraphasic mutilation of words, no errors of inflection and declension, verbal paraphasia is absent and in place of it is paralogia. The mannerisms of schizophrenic speech contradict the assumption of a disturbance of reproductivity." He discusses more in detail the theory of ellipses, derailments, paralogia, word salad and neologisms.

There have been a few other recent contributions to the subject of schizophrenic thought and speech which, as they run more in line with my own thinking on the subject, I will refer to more fully. These studies equate the processes of thinking of schizophrenic persons with those of primitive peoples and of children.

Thus Domarus[8] describes certain modes of thought which appear among primitive races and compares them with "normal," or scientific and logical, thought processes which have survived as the "fittest," that is, the most useful in dealing with external reality. He recognizes three stages in development: the prearchaic, the archaic-paralogical and

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7. Schneider, C.: Ueber die Unterschiede zwischen schizophrener Sprach und Aphasie und zur Theorie der schizophrenen Sprachstörungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **96**:251 (April) 1925.

8. Von Domarus, E.: Praelogisches Denken in der Schizophrenie, Ztschr. f. d. ges. Neurol. u. Psychiat. **87**:84 (July) 1923; abstr., J. Ment. Sc. **70**:310 (April) 1924.

the paralogical-logical. In the prearchaic, which may be supposed to have existed in such primitive forms as *Pithecanthropus* and in schizophrenic stupor, there is a generally lowered intensity of images, whether sensory or motor, and of affectivity, and a lack of any attempt to establish relationships between these images, and a lack of any real thought process. In the archaic thinking of a prelogical kind, found among primitive savage races, the vividness of the images is greater than among more highly developed races, and the effect produced in the observer is projected and believed to be an inherent attitude of the object, which thus acquires a "demonic" character. All things which arouse a similar emotion are thought of as being actually the same. In dementia praecox there is a similar loss of objectivity; hallucinations and reality are imperfectly distinguished, and every happening has a meaning and effect on the observer; the idea of an action produces the action directly, instead of offering a possibility of action, and this is interpreted as a compulsion from without. Paralogical thinking is a stage beyond this; identification of objects is based on similarities, differences being neglected. It follows the "law of participation" described by Lévy-Bruhl [9]—all things with the same qualities are the same: "certain Indians run fast," "stags run fast," therefore, "some Indians are stags." This form of thought is common in dementia praecox.

Storch, in an interesting paper,[10] takes up for consideration the ways in which the schizophrenic patient reacts to the world of reality and to his inner autistic world or world of fantasy. While for the normal person the chief criterion of the world of real objects is their independence of him, whereas imaginary things depend for their existence on him, the general characteristic of the schizophrenic patient's experience is that his mental and imaginary experiences have a substantial and concrete nature when the normal person would see only symbols and analogies. His thoughts have magic power and can produce real results; they have for him a substance and he can manipulate them physically. Speech has preserved many expressions which for the normal are symbolic, but for the patient actual; we "look down" on despised people—for the patient the physical act of looking down on some one makes him despicable; we "collect our thoughts"—the patient makes gestures with similar meaning.

With regard to the world of hallucinations he says some patients feel these to be a representation of certain aspects of their own ten-

9. White, W. A.: Review of Lévy-Bruhl's Primitive Mentality, Psychoanalyt. Rev. **11**:67 (Jan.) 1924.

10. Storch, A.: Bewusstseinsebenen und Wirklichkeitsbereiche in der Schizophrenie, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:321 (April) 1923; abstr., J. Ment. Sc. **70**:308 (April) 1924.

dencies, distinguished by their vividness and actuality from mere imaginings, existing external to themselves, but differing from other external objects in their special relation to and dependence on the observer's ego—emanations from that ego.

The patient views the world of reality as changed by his delusional ideas as a new mode of existence; it has lost its identity as something separate from him, and is experienced as a sort of concrete interpenetration. We say that some one puts his whole soul into his work—the patient experiences this as a concrete loss of a part of himself; we speak of "drinking in something with our eyes"—for the patient an actual interchange of substance occurs; every activity in relation to something of emotional interest to him is an exchange of forces between the ego and the object. This recalls the emanation idea of primitive peoples—the idea of forces which pass through space from one object to another. In a similar way there is an interweaving of the patient's personality with that of the persons about him; the feeling of "being one with somebody," which we mean symbolically, is felt as actuality by the patient; this same sense of union is felt with the universe, and is not unlike the experiences of mystics; he becomes God by this same process of interpenetration.

While his peculiar experiences have a concrete actuality for the patient, yet they are different from his experiences of the world of reality. The appearances of this autistic world are as vivid and real as external objects but their reality is different in that it depends on the observer. Attempts may be made to fit this world into external reality, such as ascribing sensations or pictures to some apparatus; or the patient may ascribe the appearance to some special gift of spiritual vision.

There are two types of the interpenetration of the self and the world; in the one the whole world may seem to be absorbed into the self, in the other the self is absorbed into the world. For the patient of the former type all objects are merely qualities of himself made concrete, emanations from himself. This view is based on the nature of his immediate knowledge and is akin to the mental processes of savages. It corresponds to the "idealistic philosophy" which regards the phenomena of the world without material existence but merely as projections of the perceiving self. For the patient of the latter type the sense of being an individual is lost, the self is felt as a dependent part of the surroundings, which can penetrate into his being. He feels his thoughts are taken from him and he is thus deprived of portions of himself; he refuses to speak, since speech is a "giving away of himself" in the most literal sense, and he withdraws from all conflict with the external world to protect himself from the sense of loss which is accompanied by tension and emotional discomfort. Impulsive actions

may be a reaction to such tension. He feels safe only in the most complete severance from the world, in the extinction of all relationships with it.

Wildermuth has studied the similarities between the real and fantasy worlds of schizophrenics and the real fantasy worlds of children.[11] Meyer-Gross had already called attention to the similarity in the behavior of the schizophrenic to that of the child at play—a behavior in which the child lives in a world of its own, but is capable at any moment of exchanging it for reality; he keeps them apart, however, and regards as a spoil-sport any one who tries to mix them.

The similarity is even more far-reaching than this author has shown. The difference between the child and the patient is mainly that the former can always return to reality, and that after puberty he ceases to be capable of living in the autistic world with the former completeness. As the child grows older he comes to require more and more reality to make his game of pretense satisfactory.

Not only does the schizophrenic's conduct strike the observer as resembling play, but he himself feels it subjectively to have a similar quality. He says people are making a game of him; it seems as if he were taking part in some play that is going on, or he plays a part without completely believing himself to be the imagined person he represents; or the hallucinations give him the impression that some sort of moving picture or theater is going on.

Many other childish manifestations resemble those of schizophrenic persons: children's jokes, tricks, and plays in words have a similar autistic character, with no apparent meaning in relation to actualities, and this changes at puberty.[12] Children, like the patients, love to make up a sort of neoplastic language of their own, having meanings known only to themselves or their immediate circle. Perseveration and stereotypy in speech and actions are often observed in children. Their musical performances show the same mechanical rendering, and the same preference for simple melodies and rhythms as are found in schizophrenic patients. The child lives far more in his unconscious or subconscious self than does the adult, and has only partially discovered himself as a personality—has not become a problem to himself. This may be due to incomplete development; the author considers the part played by defect of the ductless glands, and the concept of the imper-

11. Wildermuth, H.: Schizophrene Zeichen beim gesunden Kind., Ztschr. f. d. ges. Neurol. u. Psychiat., September, 1923; abstr., J. Ment. Sc. **70**:311 (April) 1924. In referring to this paper of Wildermuth and the two preceding papers of Storch and Domarus, I have made free to use, in considerable part, the actual language of the abstractor (M. R. Barkas) as it appeared in the Journal of Mental Science.

12. Jelliffe, S. E.: The Signs of Predementia Praecox, Am. J. M. Sc. **134**:157 (Aug.) 1907. A similar conception may be found in this reference.

fectly developed organism which fails when it has to face the tasks of adult life, and thus make it possible for mental traumas to determine the onset of the symptoms.

Schizophrenia is a regression psychosis. Without undertaking to defend this statement I will merely add that, if it is true, then we should expect to find, as indicated in the opinions just cited, that the thinking and the speech of schizophrenic patients would show characteristics of earlier stages of development, would illustrate earlier genetic levels. If this is so, and it is the way in which I view the situation, then a law of the development of language which expressed the direction in which that development proceeds would by implication indicate what we might expect to find in schizophrenic speech. Storch [13] has endeavored to formulate such a law. The change according to him is from feeling, concreteness and perception in the direction of reasoning, differentiation and abstraction.

In this connection a more recent contribution of Domarus [14] on the relation of normal to schizophrenic thinking is important. He concludes, as we should expect if it is possible to formulate a law of the development of thought such as that of Storch's and if schizophrenia is a regression psychosis, that the principles of normal thinking are sufficient to account for schizophrenic thought processes. He discusses the analogies between normal thinking and schizophrenic thinking under four heads—schematization, recognizing analogies, inferring causes and making identifications—as the psychic activities by means of which what is given in experience is elaborated. To illustrate that in schizophrenic thought the same processes are in operation the following example is cited in which it may be clearly recognized that the elements of thought which we have ourselves sought out, gathered together and unified, as it were "artificially," may be again separated "artificially"—that these elements are not unchangeable parts, but, to use *termini technici*, "divisibles."

A schizophrenic patient brought the following ideas into the closest relationship: Insane asylum—Herzberge—shark—sailor—phimosis—coffee. From her it was learned that she had abbreviated Irren-Anstalt-Herzberge to IAH by using the first letter of each word. Reversing the order of the letters this becomes Hai or shark. As the shark is represented with fins and as sailors have flaps on their caps she associated these two ideas. Now sailors have also little bands (Bändchen) on their hats and as the frenum of the prepuce is called Eichelbändchen she made the association of the sailors with phimosis (of which she had

13. Storch, A.: The Primitive Archaic Forms of Inner Experiences and Thoughts in Schizophrenia, Nerv. & Ment. Dis. Monog. series, no. 36.

14. Von Domarus, E.: Ueber die Beziehung des normalen und schizophrenen Denken, Arch. f. Psychiat. **74**:641 (Aug.) 1925.

a very naive idea) and this led her to make an identification with acorn—coffee, because the word for glans (Eichel) and acorn are the same.

This astonishing connection between ideas which for us are entirely heteronomous is due to the fact that in the ideas or words of the patient elements are sought out and distinguished as separate which would never have attracted our attention; or to use the terms of the article, these elements are schematized and brought into connection with one another, that is, regarded as analogous or even identified. Heteronomes according to the normal view become homonomes for the patient. He concludes that it would be scarcely possible to establish, from the processes of thought themselves, a criterion which should be regarded as normal in contradistinction to what should be considered schizophrenic, though it might be possible to set up such a criterion from the products of thought.

This last illustration is a good example of how far we have come since the days of the "faculty psychology," the days when the mind was supposed to be built up of elements of sensation as matter is built up of atoms. Such a way of thinking about the mind has been long since abandoned and the thought that has taken its place has found expression in the new Gestalt-Psychologie. Apropos of this matter Koffka concludes his book "The Growth of the Mind" with the following significant sentence: "The nature of mental development as it has been revealed to us is not the bringing together of separate elements, but the arousal and perfection of more and more complicated configurations in which both the phenomena of consciousness and the functions of the organism go hand in hand."

Storch, in his important work,[13] discusses schizophrenic thought from many angles, particularly: the similarity of the consciousness of the objective world and of the consciousness of self in schizophrenic patients to these structures in primitive man; the derivation of schizophrenic condensation, distortion, and formation of symbols from the manner of thinking in concrete complexes as revealed by genetic psychology; the falling apart of the ego into its component parts; the loss of the boundaries of the ego; participation; fusion of the ego with the objective world; identification; magic-taboo attitude; magic primitive transformations of the personality. He quotes at some length, as an example, the analysis by Tuczek[15] of the speech of a catatonic patient. He sums up the phenomenologic expressions of the dynamic disturbance as loss of constancy and definiteness of the structure of the object, the disappearance of the boundaries of the ego and the abolition of the consciousness of self. These phenomena and the outbreak of the magic primitive feelings and trends of experience are caused by a relaxing of the rational superstructure. The resemblances between the

15. Tuczek: Ztschr. f. d. ges. Neurol. u. Psychiat. **72**:279 (Aug.) 1921.

schizophrenic and the primitive psychic conditions may be explained from this point of view. In the dynamic explanation of these disturbances he follows Berze [16] and Kronfeld.[17] According to Berze phenomena which are regarded as expressions of a more primitive level of the ego—such as loss of completeness and constancy of consciousness of the ego; division of the ego into partial components; magic personification of components of the ego; loss of the ego through magic influences; reconstruction of the ego—are due to insufficiency of the higher functions. The ego has not had sufficient strength to maintain its integrity and falls apart as the result of the bombardment of outside forces.

The fundamental view of Kronfeld is that the archaic-primitive formations break through rents in the psychic superstructure. He says "Here and there isolated intentions are destroyed, interrupted, rent; the primitive psychic superstructure, driven upward by the original instincts, through their mechanism cause new formations to emerge in the gaps, and there now sets in a constant antagonism between the two spheres. At one time the upper sphere closes itself and becomes dominant, at another new rents are made in it." "A relaxing of the psychic apparatus in the highest strata of consciousness takes place. Through the hiatuses are thrust up the representations and projections of archaic preformations, which are driven upward out of the primitive instinctive life. These elements set up a magic, mythical, prelogical method of objectification side by side with or overshadowing the real ego which they tend to overwhelm. Thus arises the dynamism which is so characteristic of the schizophrenic psychoses in which the awful archaic instinct-ego headed for unbridled realization, ever besetting the psychological subject oriented toward reality, but now weakened and mutilated, at one moment unites with the real ego and at another separates from it—always acquiring more place, conquering position after position, and finally suppressing altogether the outer as well as the inner world of phenomena through the abundance of its own construction."

Storch goes on to express the dynamics of this conflict in words that are worth while quoting. He says: "If the crumbling of the rational superstructure is so extensive as to lead to the loss of the boundaries of the self, the inner conflict may be wrongly located beyond the personality and shifted entirely into the external world. The outer reality then becomes merely a reflection of the inner world, a symbol of the soul and its conflict. The battle of the ego, oriented toward ethical values,

16. Berze: Primäre Insuffizienz der psychischen Aktivität, Leipzig and Vienna, F. Deuticke, 1914.

17. Kronfeld: Ueber schizophrene Veränderungen des Bewusstseins der Aktivität, Ztschr. f. d. ges. Neurol. u. Psychiat. **74**:15 (Sept.) 1922.

against the repelled instinctive urges, is projected into the cosmic plan and takes the form of onslaughts of mythical powers on one another. The implacable conflict of the soul is resolved into a conflict of good and bad powers—into a struggle between the ultimate principles of the universe."

Here we have the fundamental theory of the nature of schizophrenic thinking and by implication of schizophrenic language, although we should bear in mind the modern tendency to think of language as a mode of behavior with certain purely pragmatic values of its own. It is an instrument for effecting certain social ends and needs to be considered in this aspect. As Malinowski [18] puts it, it is a mode of action rather than a countersign of thought or an instrument of reflection. He emphasizes that neither a word nor its meaning has an independent existence and says: "The Ethnographic view of language proves the principle of Symbolic Relativity as it might be called, that is that words must be treated only as symbols and that a psychology of symbolic reference must serve as the basis for all science of language. Since the whole world of 'things-to-be-expressed' changes with the level of culture, with geographical, social and economic conditions, the consequence is that the meaning of a word must be always gathered, not from a passive contemplation of this word, but from an analysis of its functions, with reference to the given culture. Each primitive or barbarous tribe, as well as each type of civilization, has its world of meanings and the whole linguistic apparatus of this people—their store of words and their type of grammar—can only be explained in connection with their mental requirements."

These hints from Malinowski I have introduced to indicate just how the meaning of a symbol must be reached from first understanding its functional significance and not from the point of view of the superstition that, in some way, words are things.

Storch starts the final section of his book very significantly with this paragraph: "From what we have here given we might seem to support the view that the schizophrenic sinks back entirely into a primitive world of feeling and thought—into the world of the percept-images, the magic wish—fulfilment and metamorphoses, and the mystical participations. That such a view is only admissible with considerable modification we have already stated at the close of Section I." If we turn to the close of Section I we shall find that he means by "modification" that, to the extent that the patient's thinking is not fully destroyed, to the extent that he retains capacity for contacting effectively with reality, to the extent that logical thought is capable of assuming the

18. Ogden and Richards: The Meaning of Meaning, New York, Harcourt, Brace & Co., Inc., 1923, supplement 1, Malinowski, B.: The Problem of Meaning in Primitive Languages.

supremacy, at times, the patient is not reduced, unqualifiedly, to a primitive level. His "modification," therefore, has only to do with the extent of the regression not with its nature. In putting it as he has I feel that he has failed to emphasize the more obvious features of the situation for our purposes, at least. I am reminded by this of the somewhat corresponding situation, on the advent of the intelligence tests, and the fact that we had to learn that a feebleminded child that measured up to, say 9 years old by the tests, could in no wise be equated with a normal boy aged 9.

Just in the same way we must be careful not to equate the regressive psychotic and the primitive too literally. That there are close analogies in their respective ways of thinking there is no doubt and that the recognition of these analogies has been of the utmost importance in enabling us to understand schizophrenic thinking must be acknowledged but perhaps, to be on the safe side, the matter should rest there for the present at least.

However, I cannot resist speculation and an attempt to formulate the analogy more satisfactorily—sufficiently so at least to assist in thinking about it. I feel that we must presuppose some relationship between the machine (the mind) that handles reality and the product (ideas, words) that the machine turns out. If we feed paper and ink into a modern Hoe press a seventy-two page folded newspaper will be the result. If we feed paper and tobacco into another kind of machine cigarets come forth. Here is a reciprocal relation between the machine and what goes into it. Not only will the Hoe press not produce cigarets if fed paper and tobacco nor the cigaret machine produce a newspaper if fed paper and ink, but the normal adult if fed reality (through the senses) will not produce schizophrenic thoughts and expressions nor will the schizophrenic produce normal ideas and speech. In the latter case it is questionable how much reality gets in, the action of the machine on the environment is selective, it picks out only those elements suited to it to elaborate into thoughts and express in speech. I like to think of regression then as a dropping back to the use of a simpler type of machine for handling reality. Reality poured into this simpler machine must result in different products than if poured into the more complex higher type of machine. This aspect of regression, the reduction to a simpler set of mechanisms with which to deal with reality, I like to call functional decerebration.

This way of thinking of the processes and content of a regressive situation is, to my mind, very helpful. However, there are other aspects which need consideration. Precisely the same analogies as have been stressed as between the regressive state of schizophrenia and the psychology of primitive man could also be formulated as between schizophrenia and the psychology of the child. I will not further discuss this analogy

except to say that the end-result in our thought and speech, whatever it may be, contains phylogenetic and ontogenetic factors inextricably mixed and that I do not believe that we can tell from the appearance of a given content to which it owes its origin. Quite probably the two are only aspects of the same reality, another example of ambivalence, and must ever appear in association and the only questions may be: Which aspect is the more stressed? or Which, for the time being, is more prominently in evidence? In any event, from a therapeutic point of view at any rate, it would be well to consider all material as ontogenetic in origin until proved otherwise. If we should approach therapeutic problems with the conviction that the whole affair was hereditary there would be little use in attempting anything. Heredity is too much the catchword for do-nothingness, and hopelessness.

Another point should be made at this time, after what has been said of the loss of the boundaries of the ego, its indefiniteness, etc. These expressions are apt to be equated, if we do not think carefully, with such concepts as disintegration and dementia. We must not lose sight of the magic of words and not be led astray by the old meanings when we are striving for new ones but are forced to use the words in current use.[19] Let me give an example. The Melanesians have special words to denote a definite number of certain objects, thus ten coconuts, ten canoes and ten fishes would each be expressed by different words. The Gestalt psychology gives the key to the explanation of this state of affairs remnants of which we have in our current language such as our use of different words to denote groups of different animals such as a flock of sheep, a pack of wolves, a herd of cattle, a bevy of larks, a covey of partridges, a shoal of fish. The Melanesian, at his level of psychologic development, applies a word to a given situation, not to some part of it. He sees the whole configuration of which the coconuts, the canoes or the fishes form but a part. He does not see the canoes, for example, in their setting, but he sees canoes and setting altogether as a unity and names that unity accordingly. Now regression to a childish or primitive level of this sort, which is what occurs in those conditions in which the ego is said to lose its clearness of definition, does not imply disintegration in the sense of disorder but regression to a different kind of order or, as my friend Korzybski would say, to a lower order of abstraction.

This is important for a principle is embodied in the nature of this change. When we advance from a lower order of abstraction, such as that of the Melanesians, to a higher order of abstraction in which we are able to use a common qualifier, ten, for each of these classes of objects, what happens is, not only have we succeeded in analyzing the

19. Ogden and Richards (footnote 18, chap. 2).

situation and separating its component parts more successfully but we have also synthetized its various parts, such as number in this instance. This means that analysis and synthesis go hand in hand, are parts of the same process, ambivalent opposites which cannot be separated one from the other.

Thus, for the Melanesians, they have analyzed their environment into its several, concrete situations. Number has not yet been analyzed. It is much later that ten, for example, comes to be something separate from the things it qualifies. It then may be applicable to anything and when we say ten it means ten of anything. Ten has thus emerged by analysis while the objects to which it is applied have flowed together into a new synthesis. Similarly, when the schizophrenic patient regresses, not only does the environment flow together into a new synthesis but also he analyzes it more completely into its separate configurations as they are each presented to him through perception. His is literally a pluralistic universe.

This principle, namely, that the schizophrenic thought processes and language are of a lower order of abstraction, accounts, in part at least, for another phenomenon. If by a process of regression the mechanisms of thinking tend to ever more primitive levels then we should expect them ultimately to arrive at a concrete perceptional level, and when this occurs hallucinations, which have long been regarded as evidence of the schizophrenic splitting, come into the picture. While I believe that there must be other factors to account for the hallucinations they are at least to be expected as the natural outcome of regression —as are the forms of thought already referred to; namely, the feeling that thoughts can be manipulated as though they had substance, and actions that indicate that expressions we regard as figurative such as "looking down" on despised people or "collecting our thoughts" are taken literally.

It comes about, therefore, that we cannot understand the language of the schizophrenic patient without the aid of these principles, because the language of a lower level of psychologic development, or a lower order of abstraction, must remain unintelligible to those who think in the terms of higher levels. The whole problem of the understanding of the psychoses, from this point of view, might be well considered as the problem of the translation of the language of the psychoses.

It would seem that for a complete understanding of schizophrenic language we should have an understanding of language itself, its origin and development. The philologists themselves, it would seem, have no satisfactory theory for the origin of speech. In the correlation of total situations as set forth in the Gestalt psychology, with the reflex sounds associated with them such as the cry of pain and the interjections such as "brrr" indicative of cold or chilliness, and the onomatopoeias

such as bang, crash, snap, fizz, etc., there is enough material, in quantity, to account for the multiplicity of words embodied in a language.[20] Here should be included the whole evocative function of speech,[18] that is, the use of speech, not primarily to express thought, but to evoke a desired state in the listener, such as the cry of the child or patient producing anxiety in mother or nurse and solicitous care. The devices of poetry suggest themselves as examples in the realm of the higher sublimations, for example, the use of the refrain in Poe's "Raven." [21] How these become combined and differentiated and how new forms arise is a question of too great magnitude to undertake here.[22] I will merely make a few suggestions.

I think in the study of speech in general and schizophrenic speech in particular the pleasure-pain principle and the dynamics of the mouth zone offer ways of approach that may well be expected to lead to important material.

In regard to the pleasure-pain principle I have in mind the ideas of Sperber [22] concerning the influence of sexual agencies in the origin and development of speech: [23] the fact that the earliest words are formed with the use of the same muscles used in nursing; that in schizophrenia there are numerous indications of pleasure in the rhythmic repetition of sounds such as in lalling, echolalia, verbigeration, perseveration and perhaps in the use of neologisms and in the use of meaningless sounds though obviously pretending to speak—pseudolalia (Stoddart).

In regard to the oral zone I have in mind the work that has been done recently in the endeavor to dismember the complexities at this level and to trace the various elements into later character qualities by

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20. Bloomfield, L.: An Introduction to the Study of Language, New York, Henry Holt & Co.

21. Poe, E. A.: The Philosophy of Composition in the Works of Edgar Allen Poe, New York, A. C. Armstrong & Son, **5**:157, 1884.

22. Sperber: Ueber den Einfluss sexueller Momente auf Entstehung der Sprache, Imago **1**:460 (Sept.) 1912; abstr., Psychoanalyt. Rev. **2**:341 (July) 1915. Berny, A.: Zur Hypothese der sexuellen Ursprungs der Sprache, Imago **2**:537 (Nov.) 1913; abstr., Psychoanalyt. Rev. **6**:99 (Jan.) 1919. Cited by Rank and Sachs: The Significance of Psychoanalysis for the Mental Sciences, Nerv. and Ment. Dis. Monog. Series, no. 23.

23. According to Sperber, for the discovery of the origin of speech it is necessary to find those typical situations which earliest brought home to man the desire to influence another person in the direction of his wishes by voluntary sounds. "Of such situations, there are only two: the child who lacks nourishment, and the sexually excited man; these two persons will perceive that the cries emitted by them, at first in purely reflex manner, call to them a person whose presence they wish, and will learn from that to repeat these cries intentionally when they want the person in question near them. From the case of the child, who calls his mother, no way leads to speech formation; easily, however, from the sexual call."

Abraham,[24] Glover [25] and Sullivan.[26] Here we find such components as the lip and facial musculature, the special senses of taste and smell, certain visceral sensations (swallowing, gastric distention) and certain respiratory elements to consider as factors. There must also be considered the nature of the stimuli to this zone during intra-uterine life and the complex, and I think very important, aspect of the embryology and phylogenesis of this whole region. In schizophrenia such symptoms as salivation, Schnauzkrampf, grimaces, respiratory crises, mutism and refusal of food attract attention to this zone very clearly. The hitching up of the various physiologic states and their component muscle tensions with the sounds that go with each should offer a field for research to identify the primitive sounds and their meanings and show how they are later combined and varied.

There is a patient in the hospital who, when she meets me, usually asks me to have "that man" dug up who is "down there" in the ground and who keeps annoying her. Aside from this delusion and its various ramifications she is clear, in good touch with reality, and a competent, in fact very efficient, housekeeper of one of the small cottages. If this delusional system could be fully analyzed it would, I have no doubt, reveal not only the mechanism of schizophrenic splitting but much of fundamental value to the sciences of both philology and psychology to say nothing of sidelights on religion and ethics. I cite this illustration and my speculations regarding it just to indicate how valuable I believe psychotic material is and to express the hope that the day is not far distant when the true importance of the mental hospital as a laboratory for the study of the mind and all things human will come to be recognized.

SUMMARY AND CONCLUSIONS

1. A complete understanding of the language of schizophrenia would imply an understanding of language in general of which schizophrenic language is only a part. This would further imply an understanding of thought in general of which language is largely an expression. Because of its extent this program is quite impossible, but certain principles need to be clearly in mind in order to avoid taking over, in any attempt to understand the language of schizophrenia, certain misconceptions in both of these territories which are still rife, not having been as yet

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24. Abraham, K.: The Influence of Oral Erotism on Character Formation, Internat. J. Psycho-Analysis **6**:247 (July) 1925.

25. Glover, E.: The Significance of the Mouth in Psycho-Analysis, Brit. J. M. Psychol. **4**:134, 1924. Notes on Oral Character Formation, Internat. J. Psycho-Analysis **6**:131 (April) 1925.

26. Sullivan, H. S.: The Oral Complex, Psychoanalyt. Rev. **12**:30 (Jan.) 1925.

fully replaced by the newer ways of thinking about the matters involved. These are, in the main, the following:

(*a*) The fact that there is no identity nor in fact similarity between an idea and its expression in speech and the object referred to. The perception red has nothing in common with a certain rate of ethereal vibrations; and while there are innumerable men in the world there is no object that corresponds to the abstraction man. Thought and its instrument language do not portray reality; they are rather ingenious dexterities that enable us to find our way about in a universe of things, events, and persons. As such they have a pragmatic justification and the symbols used remain useful and true only so long as they serve this purpose, and to the extent that they fail they lose not only their usefulness but their truth.

(*b*) Therefore, the meaning of any given expression cannot be known except as it is studied in its setting and from an analysis of its functions.

(*c*) Neither thought nor language has grown up by a coming together of elements in the manner of the synthesis of matter by atoms and molecules. The old "faculty psychology" needs to be abandoned in all its implications. The earliest perceptions were not built up of sensations but the entire situation, configuration, in the sense of the Gestalt psychology, was perceived as a totality.

(*d*) Language has certain functions in its own right aside from being a means for the expression of thought. Here we have certain practically reflex phenomena, which seem to be most closely related to certain psychologic states such as the cry of pain and such interjections as "brrr" indicative of cold or chilliness. Here should also be included the whole evocative function of speech, that is, the use of speech, not primarily to express thought, but to evoke a desired state in the listener. The devices of poetry suggest themselves as examples in the realm of the higher sublimations.

There is one psychiatric assumption I have made and which is fundamental to my approach to the problem of the language of schizophrenia. It is that schizophrenia is a regression psychosis. This is of the greatest importance for, if it is true, we should expect to find in the thinking and in the language of schizophrenic patients characteristics of earlier stages of development, earlier genetic levels.

3. The development or evolution of thought and speech, the assumption of genetic levels, implies that there must be a law in accordance with which this development proceeds. This law is that thought and language in their development change from feeling, concreteness and perception in the direction of reasoning, differentiation and abstraction.

4. The law of schizophrenic thought and language must be the reverse of the law for their development, on the assumption that schizophrenia is a regression psychosis.

5. This reversal of the law of development implies results that are very different from those implied in the old terms "disintegration" and "dementia."

6. This reversal can be briefly and simply indicated. The language of schizophrenia is of a lower order of abstraction than normal adult language.

Example.—The women patients of Bertschinger who recovered following extraction of teeth equated this operation with childbirth. This equating of all things that come out is on all fours with the childish way of thinking of the little girl who divided all animals into two classes: first, horses, including all four-footed animals, even a tortoise, and second, fishes, including all that moved without the use of feet, for example, birds and flies. It is similar too to the practice of certain people who, when a woman is about to be confined, see that she has no knots on her garments. These are all untied to facilitate delivery.

7. The thinking and the speech of schizophrenia while of a lower order of abstraction nevertheless make use of words which we are accustomed to use to express a higher order. This discrepancy is one reason why such language is so hard for us to understand. Another reason for our difficulty in understanding the schizophrenic patient is that while some of his symbols are of a lower order of abstraction by no means all of them are so that there is a strange mixture which further confuses our understanding. Still another difficulty is due to the magic of words. We are still far from free of this influence and are therefore forced to think that when there is a word there must be a thing corresponding to it and also forced to think of the wording as necessarily meaning what it usually has meant in our experience.

8. The reversal of the law of development in schizophrenia also accounts, in part at least, for hallucinations which have long been regarded as signs of the schizophrenic splitting. Regression must lead ultimately to concrete, perceptional configurations and all that that implies.

9. In this degradation of speech to a lower level of abstraction it must be remembered that analysis and synthesis are ambivalent opposites which do not exist at opposite poles but go along side by side—the parallel path of opposites. Every analysis means a synthesis—every synthesis means an analysis.

10. The pleasure-pain principle needs to be considered in the understanding of certain forms of schizophrenic speech, especially those forms showing rhythmic repetition of sounds such as lalling, echolalia, verbigeration, perseveration, etc.

11. The study of the oral zone is suggested as offering possibilities for understanding these pleasure-pain components of speech and perhaps other more subtle elements.

12. For the understanding of the language of schizophrenia, therefore, the whole dynamic situation needs to be comprehended. The main obstacle to this understanding has been, in the past, the magic of words.

13. The study of sounds emitted in connection with certain physiologic states with their component muscular tensions is also suggested as a means of getting at the primitive components of speech which later are combined, recombined and varied in innumerable ways.

THE BLOOD SUGAR CURVE IN MENTAL DISEASE

II. THE SCHIZOPHRENIC (DEMENTIA PRAECOX) GROUPS *

JACOB KASANIN, M.D.
BOSTON

In a previous communication the results of a study of the blood sugar curve in epidemic encephalitis [1] have been reported. The aim of the present article is to discuss in a similar manner the significance of the sugar curve in cases belonging to the schizophrenic group (dementia praecox). In these cases a tendency toward a low metabolic rate has been demonstrated.[2] The study of the sugar curve was undertaken to see if the low basal metabolism was reflected in the sugar metabolism as judged by the glucose tolerance tests. The correlation that Janney and Isaacson [3] have found between these two factors in hyperthyroidism and hypothyroidism has not been possible in this study.

All the curves studied in this laboratory have been carried out according to the technic of Janney and Isaacson. The blood sugar determination was done according to the method of Folin and Wu.[4] We have used the classification of Mosenthal [5] to designate the curves, largely founded on Gray's [6] work: (1) the normal curve, which shows a rise during the first hour not exceeding 160 mg. per hundred cubic centimeters and returns to the normal fasting level (between 60 and 120 mg. per hundred cubic centimeters) at the end of the second hour after ingestion of glucose; (2) high curve, which shows a rise during the first hour exceeding 160 mg.; (3) low curve, in which at no time during the period of observation the blood sugar rises above 120 mg. per hundred cubic centimeters.

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* From the Laboratory of Internal Medicine in the Boston Psychopathic Hospital.

1. Kasanin, Jacob; and Grabfield, G. P.: Blood Sugar Curves in Epidemic Encephalitis, Arch. Int. Med. **37**:102 (Jan.) 1926.

2. Bowman, K. M., and Grabfield, G. P.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **9**:358 (March) 1923. Bowman, K. M.; Eidson, J. P., and Burladge, S. P.: Biochemical Studies in Ten Cases of Dementia Praecox, Boston M. & S. J. **187**:358 (Sept. 7) 1922.

3. Janney, N. W., and Isaacson, V. I.: A Blood Sugar Tolerance Test, J. A. M. A. **70**:1131 (April 20) 1918. Olmstead, W. H., and Gay, L. P.: Study of Blood Sugar Curves Following a Standardized Glucose Meal, Arch. Int. Med. **29**: 384 (March) 1922. Janney, N. W., and Henderson, H. E.: Concerning the Diagnosis and Treatment of Hypothyroidism, Arch. Int. Med. **26**:297 (Sept.) 1920.

4. Folin, O., and Wu, H.: J. Biol. Chem. **38**:81 (May) 1919.

5. Mosenthal, H. O.: The Interpretation of Sugar Tolerance Tests, M. Clin. N. Amer. **9**:549 (Nov.) 1925.

6. Gray, H.: Blood Sugar Standards, Arch. Int. Med. **31**:243 (Feb.) 1923.

We have studied by this method forty cases,[7] of which seven were eliminated on account of various extraneous factors which might modify the sugar curve. In W. Z., there was a definite subacute arthritis which, according to the investigations of Pemberton and Foster,[8] tends to elevate the sugar curve. In A. S., there was a recent, second degree burn, and the absorption of toxic products might change the form of the sugar curve. In another case, subacute ulcerative endocarditis with active joint disease and leukocytosis was present. The mental symptoms were of the hebephrenic type, but the presence of fever may change the sugar curve. In E. D., hypertension and arteriosclerosis were factors which may alter the sugar curve. In A. K., the infantile uterus, cessation of menstruation four years before admission to the hospital and obesity suggest an obvious endocrinopathy. In the sixth case there was an enlarged thyroid, rapid pulse and a fine tremor, but no other signs of hyperthyroidism. The seventh case had to be eliminated on account of an active tuberculous process in the lungs. None of the remaining thirty-three cases showed evidences of definite endocrinopathy. The blood Wassermann test was negative in all cases.

Of the thirty-three patients examined, eleven had a normal sugar curve. The remainder showed an abnormal curve according to Mosenthal's criteria. Thus, two thirds of our cases show evidences of derangement in the carbohydrate metabolism, but this does not seem to be due to any single specific physiologic or pathologic process as far as we were able to ascertain. Since many organs may be responsible for the variations in the carbohydrate metabolism, it becomes difficult to evaluate the significance of the high percentage of abnormal sugar curves in these patients. When we compare the cases with a normal sugar curve to those with an abnormal sugar curve there seems to be no great difference in the clinical pictures presented.

In table 1, we have collected the cases with an "abnormal" sugar curve. "High" sustained sugar curves have been reported by numerous observers in catatonic stupors. In our series, the two highest sustained curves were obtained in patients who at the time were in a stupor (cases 3 and 15). On the other hand, a perfectly normal sugar curve was obtained in patient 29 who was, and still is, in a stupor. In table 1 there are nine patients who had a high sugar curve. Of these, only three were in a catatonic stupor when the sugar curve was done (cases 3, 4 and 15). Two cases were convalescent following a catatonic stupor (cases 18 and 19), while the remaining four high curves were

7. Abstracts of the case reports have been printed in the author's reprints, a copy of which will be furnished by him on request.

8. Pemberton, Ralph; and Foster, G. L.: Study of Arthritis in the Army Based on Four Hundred Cases, Arch. Int. Med. **25**:243 (March) 1920.

obtained in cases in which the clinical picture consisted in tremendous development of fantasy at the expense of reality, with occasional appearance of hallucinations. The large incidence of cases with a high sugar curve in stuporous conditions does not seem to be limited to schizophrenic stupors only; thus, in very profound depression, when a person is overwhelmed by grief, we also get a high sustained curve, as will be reported in a later communication. It seems safe to conclude that in psychogenic stupors one frequently encounters a decreased carbohydrate tolerance. That this mechanism may be biologically protective to the person in

TABLE 1.—*Twenty-Two Cases with Abnormal Sugar Curves*

						Blood Sugar in Mg. per 100 Cc.						
No.	Initials	Date	Sex	Age	Weight in Pounds	Fasting	First Hour	Second Hour	Third Hour	Blood Pressure	Urine	Basal Metabolism
1	S. P.	8/31/23	F	22	113	98	124	109	100	130/78	Negative	— 5%
		9/ 5/23	..	..	...	87	98	95	64			
		9/10/23	..	..	...	80	106	94	92			
2	A. B.	11/30/23	F	27	190	67*	85	87	144	130/80	Negative	.....
3	M. M.	8/30/22	F	19	175	119	190	195	...	138/76	Negative	—10%
4	P. S.	10/ 2/24	M	22	136	97	...	187	...	130/100	Negative	— 7%
5	G. B.	10/ 6/24	F	25	127	78	171	83	60	100/70	Negative	— 2%
6	C. C.	6/26/23	F	26	150	149	190	...	125	115/85	Albumin, slight trace	.....
7	M. C.	.....	F	21	123	93	168	109	83	118/72	Negative	—22%
8	A. L.	11/26/24	M	39	129	81	77	85	80	135/85	Negative	.....
9	H. J.	9/12/25	M	28	146	109	104	85	92	135/85	Negative	.....
10	I. G.	12/18/24	M	30	127	83	58	88	85	120/75	Albumin, slightest possible trace	.....
11	D. R.	4/ 8/25	M	33	130	91	105	71	75	120/70	Sugar, trace	— 4%
12	O. D.	2/18/25	F	14	110	82	99	94	...	110/80	Sugar, trace	—29%
		5/29/25	Patient improving			102	148	154	91			
13	M. D.	4/ 6/25	F	36	96	82	110	82	67	130/80	Negative	+ 4%
14	W. B.	10/ 1/24	M	22	165	70	161	92	123	160/90	Negative	— 1%
15	A. S.	6/19/25	F	36	140	97	236	159	145	120/80	Negative	.....
16	E. C.	6/24/25	M	47	115	106	112	123	71	105/75	Negative	.....
17	D. S.	12/19/24	M	32	126	77	113	95	133	120/70	Negative	+ 5%
18	M. J.	5/19/25	F	18	135	96	185	132	127	120/80	Negative	— 9%
19	J. L.	6/24/25	M	21	100	103	174	125	74	105/65	Negative	.....
20	G. S.	6/22/24	F	27	123	99	183	179	137	130/80	Negative	+ 3%
21	M. M.	9/26/22	F	16	110	133	152	135	101	125/95	Negative	—18%
		8/15/22	..	..	...	121	117	113	107	118/72		
22	C. W.	11/15/23	F	31	125	78*	163	142	101	120/80	Negative	.....

* Blood clotted.

stupor comes to mind as a possible explanation of these findings. Seven cases had a low sugar curve. The low sugar curve does not seem to be associated with any special clinical picture.

In table 2 are shown eleven normal curves; and review of the patients' histories shows that we are not dealing with different types of disease from the cases with an abnormal sugar curve.

Of the cases shown in table 1, three of the twenty-two made a mental recovery, while two cases of eleven with a normal sugar curve made a similar recovery; in our cases, therefore, a somewhat higher ratio of recovery is seen in the group with a normal sugar curve.

The number of cases recovered in each group is so small that it is not possible to reach definite conclusions as to the prognostic significance of a normal sugar curve.

Raphael and Parsons,[9] studying ten cases diagnosed "dementia praecox of the acute type," found a great variation in the highest level, while the shape of all the curves was practically the same. They state that in their cases the general shape of the curve differs from the normal in that the initial fasting level is lower, the acme is relatively high, and the return to the primary level takes more than three hours. In none of the cases was there noted any special physical or neurologic abnormality.

Kooy[10] made a detailed study of ten cases of dementia praecox of the hebephrenic type showing the following clinical symptoms: "Autism, loss of interest in matters of daily life, high degree of suggestibility on

TABLE 2.—*Eleven Cases of Schizophrenic Patients with Normal Sugar Curves*

						Blood Sugar in Mg. per 100 Cc.						
No.	Initials	Date	Sex	Age	Weight in Pounds	Fasting	First Hour	Second Hour	Third Hour	Blood Pressure	Urine	Basal Metabolism
23	L. B.	9/25/23	F	21	108	97	140	120	103	130/68	Negative	.....
24	L. B.	11/18/24	M	19	101	101	156	114	104	118/70	Negative	—17%
25	E. S.	11/ 4/24	M	19	124	93	148	123	95	115/75	Negative	—20%
26	M. S.	5/27/24	F	20	122	81	157	142	89	125/100	Negative	+ 7%
27	M. A.	5/19/24	F	28	123	108	152	106	94	125/100	Negative	.....
28	N. S.	12/10/22	M	15	131	114	122	96	104	110/95	Negative	.....
29	E. C.	4/ 7/25	F	22	130	88	130	120	66	95/70	Negative	— 5%
30	P. S.	5/15/25	F	16	102	89	141	138	102	106/58	Negative	+ 1%
31	A. H.	6/22/24	F	36	127	106	146	118	100	125/80	Negative	+ 3%
32	A. H.	6/22/24	F	28	77	96	160	112	89	110/80	Negative	.....
33	W. G.	6/20/24	M	33	135	100	148	111	107	120/80	Negative	—11%

the one side and of negativism on the other with all the abnormal movements and positions dependent on it, as well as the incomprehensible association in speaking and acting." In these cases he finds a constant hyperglycemic curve.

Drury and Farron-Ridge[11] studied eighteen cases of dementia praecox, delimiting the disease according to Kraepelin's classification. They obtained "a profusion of different types of curves making analysis extraordinarily difficult." They note four points of interest: "1. Acute cases give high and rather broad curves suggesting the confusional type. 2. Female acute cases give higher curves than acute male cases. 3. Chronic cases tend to give small, low curves. 4. Again the male

9. Raphael, T., and Parsons, J. P.: Blood Sugar Studies in Dementia Praecox and Manic Depressive Insanity, Arch. Neurol. & Psychiat. **5**:687 (June) 1921.

10. Kooy, F. H.: Hyperglycemia in Mental Disorders, Brain **42**:214 (Oct.) 1919.

11. Drury, K. K., and Farron-Ridge, C.: Some Observations of the Types of Blood Sugar Curves Found in Different Forms of Insanity, J. Ment. Sc. **71**:8 (Jan.) 1925.

chronic cases are lower than the female." Drury and Farron-Ridge say nothing about the physical condition of the patients.

Bowman, Eidson and Burladge [2] find a tendency toward a sustained blood sugar curve in a study of ten cases of dementia praecox exhibiting no physical disorders, although a few cases showed some evidence of possible endocrine disturbance.

Lorenz [12] states that active cases of catatonic dementia praecox responded to glucose feeding with hyperglycemia in a manner that resembles the response obtained in hyperthyroidism, while several cases of simple, deteriorating dementia praecox responded to glucose feeding in a manner which resembles the response obtained in certain endocrine

TABLE 3.—*Average Curves Collected from the Literature*

Investigators	Number of Cases Studied	Test Meal	Method of Glucose Determination	Sugar Curve			
				Fasting Sugar	First Hour	Second Hour	Third Hour
Kooy	10	100 Gm. of bread and butter, 200 cc. of milk	Bang	93	123	110	110
Raphael and Parsons	11	Janney and Isaacson	Lewis and Benedict	89	145	126	92
Lorenz	54	Janney and Isaacson	Lewis and Benedict	120	160	168	124
Barret and Spirre	30	Janney and Isaacson	Folin and Wu	109	147	129	102
Henry and Mangan	9	Janney and Isaacson	Folin and Wu	105	266	198	171
Kasanin	40	Janney and Isaacson	Folin and Wu	97	146	121	102
	154	Average curve*		94	156	142	112

* The glucose meals given make the curves comparable according to Gray (Blood Sugar Standards, Arch. Int. Med. **31**: 243 [Feb.] 1923).

disorders such as dyspituitarism. He states that some of his cases responded to sugar feeding within a normal range of variation. He does not speak of the physical condition of the patients.

Barret and Spirre,[13] as a result of study of thirty cases, found variations in the sugar curve in different determinations in the same person, while the clinical condition remained essentially unaltered. They refuse to draw definite conclusions on account of the presence of other factors which may modify the sugar curve such as age, sex, weight or duration of illness. They give no data as to the physical or endocrinologic factors involved.

Henry and Mangan [14] studied nine cases diagnosed as dementia praecox in the acute stage. The sugar curves obtained are similar to

12. Lorenz, W. F.: Sugar Tolerance in Dementia Praecox and Other Mental States, Arch. Neurol. & Psychiat. **8**:184 (Aug.) 1922.

13. Barret, T. B., and Spirre, P.: Blood Analysis and Sugar Tolerance in Mental Disease, J. Nerv. & Ment. Dis. **59**:561, 1924.

14. Henry, G. W., and Mangan, Elizabeth: Blood in Personality Disorders, Arch. Neurol. & Psychiat. **13**:743 (June) 1925.

those obtained by Lorenz and by Raphael and Parsons. They find that "the glucose tolerance curves found in depression and in acute cases of dementia praecox indicate a definite retardation in glucose metabolism." They do not give the age of the patients nor their physical status.

In table 3 we have collected 154 cases from the literature. The average curve of these cases falls well within the normal limits defined by Gray.[6] In spite of some difference in the test meals given and in the technic of sugar determination, these data seem to be comparable. Although the average curves collected from the literature show no deviation from the normal, almost all observers agree that the frequency of abnormal sugar curves in dementia praecox exceeds the 17 per cent deviation which may be found in normal persons.[6]

From this compilation it is evident that we could find no curve that is characteristic of the "dementia praecox" group as a whole. This is not unexpected in view of the heterogeneous character of the clinical conditions described under this head. Patients in a stupor give fairly consistent response to carbohydrate feeding, but this does not seem to be limited to the stupors found in this disease.

SUMMARY

1. Determinations of the blood sugar curve were made according to the technic of Janney and Isaacson in forty schizophrenic cases. Of these, seven cases were discarded on account of the presence of somatic factors that might influence the curve.

2. Blood sugar curves of 154 patients of similar types have been collected from the literature.

3. The average curve, of the patients recorded in both the literature and this series, falls well within normal limits, although the percentage of abnormal sugar curves is much higher than in healthy subjects. The findings of the various authors were not consistent with each other.

4. In our cases no sugar curve characteristic of this group of conditions was found. Our results fail to confirm the observations of those who claim to have found a characteristic curve in the group.

5. Patients in a stupor usually respond with a high sustained sugar curve, although normal sugar curves, as well, may be found in stuporous patients.

ROENTGENOLOGIC EXPLORATION OF THE CENTRAL NERVOUS SYSTEM WITH IODIZED OIL (LIPIODOL) *

PROF. J. A. SICARD
PARIS, FRANCE
AND
JACQUES FORESTIER, M.D.
AIX-LES-BAINS, FRANCE

From the beginning of our experiments on the roentgenologic use of iodized oil, we have thought that this substance would be suitable for the exploration of the cavities of the spine and the skull. In spite of the remarkable sensitiveness of the nervous organs, we trusted the wonderful innocuousness of lipiodol, and the experience of four years has demonstrated the possibilities of this new method of exploration.

Why is lipiodol so well tolerated in the cerebrospinal fluid when very small quantities of mercury or arsenic give such violent reactions? The answer is simple. Lipiodol is not an oily solution of iodine, but a chemical compound in which 40 per cent iodine is firmly bound to a vegetable oil, poppy-seed oil. On account of the high percentage of iodine the opacity to roentgen rays is very great. But as the iodine is not free and cannot be detected by ordinary chemical reactives, and the absorption of the compound is very slow, all the drawbacks of iodine, its causticity and toxicity, are nearly completely cancelled. This explains why 2 cc. of lipiodol which represents 1.08 Gm. of iodine may be injected into the cerebrospinal fluid with only a very mild reaction, while 10 mg. of neo-arsphenamine can hardly be borne. But operators are strictly warned to use only fresh, pure and transparent lipiodol. Every sample of brownish color or dim appearance must be discarded. The best lipiodol is delivered in small aluminum flasks containing 5 cc.

We shall describe separately the explorations of the epidural space, the spinal subarachnoid space and the cerebral ventricles.

EPIDURAL SPACE

From atlas to sacrum, the epidural space surrounds the spinal dura and shelters it from contact with the osseous and ligamentous walls of the spinal canal. It is filled with a layer of fat, which is a protective against shocks and wounds. It is early involved in some vertebral diseases, so that its study is of real interest.[1]

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* A lecture delivered by Dr. Forestier at various institutions in the United States.

1. Forestier, Jacques: Le trou de conjugaison vertébral, Thèse de Paris, 1922.

Technic.—The epidural space may be reached by injection either into its lowest part, at the extremity of the sacral canal (Sicard's sacrococcygeal injection), or into other segments of the spine. In the latter case the technic and instruments are the same as for a lumbar puncture, but it is important that the point of the needle after penetrating the ligamentum flavum should not perforate the dura. To have some control as to the right position of the needle, 1 or 2 cc. of the novocaine or a sodium chloride isotonic solution must be quickly injected; if the injection is easy and the injected fluid makes an immediate reflux (as a result of the elasticity of the dura), it means that lipiodol may be safely injected.

Fig. 1.—Epidural injection. Normal space; sacrococcygeal route; knee-chest position during one hour; lipiodol has progressed along the fat layer of the space and partly escaped through the lumbar and sacral foramina along the nerves.

The proper exploration dose varies from 3 to 6 cc. This injection is possible at the lower part of the cervical region, and along the lumbar segment. The patient should recline on one side with the body half bent. If the dura has been perforated, it is better to try another space or delay the exploration to another day.

After the injection, lipiodol follows the action of gravity and glides along the space. When the injection has been performed above the segment under investigation, the patient should remain in a sitting position for three or four hours. On the other hand, if the injection has been made by the sacrococcygeal route, or below the segment under examination, the patient should be put in either the Trendelenburg or the knee

chest position for one or two hours; then he should lie flat for three or four hours more.

About six to ten hours after the injection, the progress of the oil stops. The substance is fixed to the fatty globules of the space, and there will be no further change in the pictures until entire absorption.

Results.—In normal subjects the track of the lipiodol along the space has the appearance of irregular, elongated splotches of ink. Its length depends on the quantity of oil injected; with 4 cc. it varies from four to six vertebral bodies. Generally, a part of the lipiodol escapes from the vertebral canal through the intervertebral foramina and is visible alongside the ribs, or the lumbar and sacral spinal roots. This happens mostly when the oil has been warmed previous to injection, and when the patient has been lying in lateral decubitus.

The characteristic pictures of pathologic conditions consist essentially in the sudden stoppage of the track, and the accumulation of the splotches against the obstacle to its progress.

Destructive affections of the spine generally cause early changes in the epidural space and these changes may be radiologically revealed by the epidural injection of lipiodol. In the case of the evolution of a primary or metastatic cancer in the spine, the blocking of the space is complete and quite characteristic. In Pott's disease, even without paraplegia, the stoppage of the lipiodol at some distance from the bony lesion indicates the limits of the tuberculous pachymeningitis. In some cases it is possible accurately to outline the height of the compression by a double epidural injection above and below the lesion. The stoppage of lipiodol is also complete in fractures of the spine with or without dislocation. Among abnormalities of the spine, spina bifida occulta sometimes stops the lipiodol as Andre Léri (Paris) has shown; this may be an indication for operation.

On the contrary, negative findings, that is to say, free epidural space, are seen in spinal rheumatism, chronic spondylosis, scoliosis and neuralgias of vertebral origin. It must be noted that the epidural space generally remains free in the case of intradural tumors, even with subarachnoid block and complete paraplegia.

SPINAL SUBARACHNOID SPACE

Exploration of the spinal subarachnoid space, with the exploration of the bronchial tubes, is the best application of the lipiodol method. It has rendered possible an early diagnosis and accurate localization of spinal cord compressions, and so has widely increased the percentage of successful operations in these dangerous affections. Ayer's method of combined cistern and lumbar puncture and the Queckenstedt test have led to great improvement in the diagnosis of spinal subarachnoid

block, but while they diagnose an obstruction, they generally fail in localizing it. Every neurologist knows how difficult the localization of the compression may be through clinical symptoms only.

On the other hand, in the course of spinal cord compressions, lipiodol may localize the obstruction with full accuracy at a period when objective sensory symptoms have not yet appeared. When these are present, lipiodol gives a localization in reference to the spine, whereas the sensory symptoms give it in reference to the spinal cord; the former indication is of much more practical value for the surgeon.

The principle of the method is the following: A large drop of lipiodol is injected into one extremity of the spinal canal, through cistern or sacrolumbar puncture. In the former case the patient must be in a sedentary or semi-recumbent position; in the latter, he must lie in Trendelenburg's position; in both the action of gravity makes the drop sink along the cerebrospinal canal down to the opposite extremity.

If the drop encounters an obstruction, it stops and demarcates its upper (cistern injection) or lower (sacrolumbar injection) limit. We must say immediately that the technic with cistern or cervical puncture is much safer, and we are grateful to Dr. J. B. Ayer, inventor of the cistern puncture, and to Dr. H. T. Patrick who told us of it while visiting our hospital department in Paris.

Technic.—1. Cistern Injection. The process is the same as that of cistern puncture, but there are a few points which must be borne in mind:

a. The patient must be in a sedentary or semi-recumbent position; while performing the puncture, the operator will take care to keep the needle in a very slightly ascending position. In case of great obliquity the needle may penetrate the cistern between the two tonsils of the cerebellum, and the lipiodol injected into this narrow space, will not sink properly into the cistern, but will stagnate, retained by surface tension.

b. A few drops of cerebrospinal fluid only should be allowed to flow out, so as to keep the dural sac as widely expanded as possible.

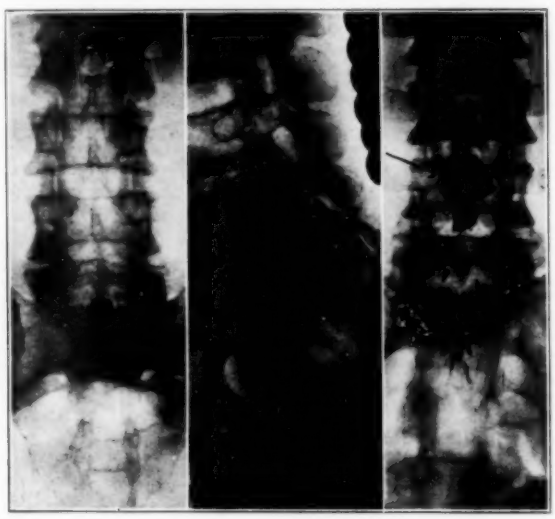
c. The dose of lipiodol should be between 1 and 1.5 cc.; the oil must be deprived of air bubbles which would disturb the sinking of the lipiodol.

d. After the injection the patient must remain for half an hour in the sedentary position and then roentgenograms should be taken in the vertical position. This is most important. Sometimes, it is necessary to take several films in different positions. In any case, several films must be taken: one of the cervico-occipital region to see whether the injection has been correctly performed; a second at the opposite extremity of the spine to see whether the globule of lipiodol has entirely

reached the sacral region; if it has not, a roentgenogram must be taken of the segment at which the stoppage has taken place.

If the roentgenologist wishes to save time and films, it will be found convenient to begin the exploration by fluoroscopy which readily shows the globule of lipiodol. But when its location has been found, it is necessary to take a roentgenogram.

In addition, the injection is equally possible between the atlas and axis, or between the axis and the third cervical vertebra. But the technic is not as safe and there may be some mistakes in cases of obstruction of the upper cervical segment of the spine.



A B C

Fig. 2.—Subarachnoid space. Normal; intracisternal injection of 1.5 cc. Roentgenograms of the same subject in three positions: *A*, lying on the back (sitting or standing position would give about the same picture); *B*, lying on the right side; *C*, lying face downward, the lipiodol has left the sacral region and is seen in the lowest part of the lumbar curvature.

2. Lumbar Injection.—Here also, the process is the same as that of lumbar puncture, except that it should be performed if possible between the fifth lumbar and the first sacral vertebra. The dose of lipiodol is the same as that above; the patient may be either sitting or lying during the injection, but the best technic is to perform the injection with the patient lying on a roentgenologic table, which allows reclining positions. The patient is firmly tied to the table and the pelvis is raised higher than the head, to an inclination of from 45 to 60 degrees or more. The slow progression of the lipiodol owing to gravity toward the cervical segment and the skull should be watched by fluoroscopy, and a film taken in this position when there is a stoppage.

The drawback in this latter method lies in the fact that many patients cannot stand this reclining position for more than ten or twenty minutes; and if the lipiodol has shown a stoppage, it will not remain against the obstacle, but will fall down to the lumbosacral angle as soon as the patient gets up again.

Reactions after the Injections.—There is no immediate reaction to the lipiodol injection; it is quite painless, and it is a matter of wonder to watch on the screen the progress of the oil globules among the roots and along the spinal cord, while the patient does not experience the least sensation. Even if, after lumbar injection, the patient is placed in a head down position and the lipiodol penetrates the cerebral ventricles, no clinical symptoms can be noticed from the passage through the cerebral foramina.

The average secondary reaction consists in headache beginning about six hours after the injection, a little rise in temperature which does not exceed one degree and sometimes some stiffness in the back; all these symptoms disappear in one or two days.

In tabes and chronic syphilitic meningitis, the reactions are generally of higher intensity; the headache is greater, some lightning pains may appear during two or three days. In a case of spastic paraplegia, we have noticed transitory retention of urine. But even then, the symptoms did not persist more than a few days. Later, the presence of the lipiodol is quite latent.

The daily examination of the cerebrospinal fluid after the injection of lipiodol shows the following data: On the second day there is a high cellular reaction, such as happens after the injection of any foreign substance into the cavity; the fluid remains clear, the protein unchanged, but from 100 to 200 polymorphonuclear cells may be counted. This reaction does not last. On the third day the number of cells drops considerably, and one week later, no positive change can be demonstrated in the cerebrospinal fluid.

Accidents.—The accidents with cistern injection are exactly the same as those of cistern puncture. The injection of 1 or 2 cc. of lipiodol is quite harmless. In our personal statistics, including over 500 explorations, we have had no fatal accidents. But we have taken great interest in the possibility of remote accidents arising from the slow elimination of the lipiodol. After four years' experience, during which we have followed the patients who had received intraspinal injections and had been operated on, we have not been able to detect one case in which the patient complained of any symptom from the presence of a small quantity of lipiodol, and no case has been reported in the literature until the

last few months. Maclaire and Sharpe [2] have reported two very similar cases of aggravation of symptoms after injections of lipiodol into the subarachnoid space for the diagnosis of spinal cord compression following a fracture in the spine. But in both cases, two successive operations had been performed after the lipiodol test, and there is no evidence that the deleterious results should be attributed to the oil.[3] It seems, however, desirable that the surgeon should remove the lipiodol during the operation whenever it is possible. This technic would prevent irritation of the meninges, if these are sensitized by the operation.

Indications.—The lipiodol test of the subarachnoid space, in spite of its harmlessness, is indicated only when partial or complete compression of the spinal cord or roots is suspected from the clinical facts and examination of the cerebrospinal fluid. It seems especially useful for the localization of tumors at the level of the conus, epiconus and cauda equina, where mistakes are more numerous than at any other level. A positive result with the Queckenstedt test, a great difference in the composition of the cerebrospinal fluid in the cisterna magna and the lumbar culdesac after Ayer's combined puncture, and last the presence of the albumino-cytologic dissociation of Sicard and Foix (increase of albumin without cellular reaction), or still more a yellow fluid after lumbar puncture, are definite indications for the lipiodol test. It is important that it be performed not less than ten days after any lumbar puncture. When an operation is to be performed, the best period seems to be from one week to a fortnight after the test.

Interpretation.—1. After intracisternal injection: From the progress of the lipiodol into the spinal canal the cases may be classified into three groups: A, the lipiodol has sunk completely down to the lumbosacral culdesac; B, it has partly stopped in its progress; C, it has entirely stopped at a determined level in the spine.

A. Patent Canal. This is, of course, normal. The shape of the globule in the lumbosacral culdesac is different according to the position of the patient. If he is sitting or standing, it looks like a radish at the level of the first and second sacral vertebra; if he is lying on the back, it is about the same. But if the patient lies on one side, the lipiodol takes the shape of a long comma behind the first three or four lumbar vertebrae; and finally, if the patient lies on his chin, there is a square globule at the level of the third lumbar vertebra. These pictures are

2. Maclaire, A. S.: Lipiodol in Neuro-Surgery, Am. J. M. Sc. **170**:874 (Dec.) 1925. Sharpe, William; and Peterson, C. A.: The Danger in the Use of Lipiodol in the Diagnosis of Obstructive Lesions of the Spinal Canal, Ann. Surg. **83**:110 (Jan.) 1926.

3. On inquiry it appears that the two reports referred to in footnote 2 were of one and the same case, though made independently.

determined by the mobility of the oil in the cerebrospinal fluid, and the curvature of the lumbar spine.

In normal subjects, it may happen that some little drops in their progress hang to a root or to some roughening in the dural wall. This must not be considered as a pathologic change.

B. Partial Stoppage. Two kinds of stoppage are not pathologic: 1. Stoppage at the point of injection; the latter may not have been performed in the right way. The most characteristic example is when the needle is inserted too deeply and too obliquely during intracisternal injection, and the greater part of the lipiodol is caught between the tonsils of the cerebellum. The black spot is then seen best in lateral view, just above the occipital bone; in some cases this abnormal stoppage is total. This incident happens very seldom and is not dangerous; it requires a second injection, a little lower, and that is all. 2. Sometimes some big drops of lipiodol stop between the seventh cervical and the sixth thoracic vertebra in quite normal subjects. This part of the canal is anatomically very narrow as we have shown. In such cases the drops are irregular, and their lower limit is generally tapered and quite different from that of the pathologic stoppages. This accident occurs generally when the cistern injection has been performed too soon after a lumbar puncture. The leakage of the cerebrospinal fluid which follows the puncture renders the dural sac insufficiently expanded, and the lipiodol may be abnormally, though not pathologically, stopped. This is why operators are warned not to perform cistern injection for roentgen-ray exploration within less than ten days after a lumbar puncture. In all nonpathologic stoppages, several radiograms taken a few days later show changes in the pictures; this is contrary to what is usually observed when there is a pathologic condition.

The pathologic conditions which may give a partial block of the subarachnoid are the following: (*a*) Chronic meningitis, generally of syphilitic origin. The drops of lipoidol are caught by the networks of arachnitis. (*b*) Chronic pachymeningitis. The shape of the stoppage is sometimes that of a comb, the teeth of which are directed downward. (*c*) Purulent epiduritis. In one such case, with abscesses in the spinal canal, we saw partial blockage of the subarachnoid space; after a successful operation a new injection of lipiodol proved the patency of the space. (*d*) At the very beginning of extradural or intradural compressions. Those cases are of delicate interpretation and an operative determination cannot yet be based on such a partial blockage. (*e*) Some intramedullary tumors with partial block give a quite characteristic picture; while a part of the lipiodol has dropped down, the remainder sticks to both sides of the canal and is divided into several strips, following one another in a line, and outlining the fusiform shape of the involved segment of the spinal cord.

C. Complete Stoppage.—1. The most characteristic pictures are given by intradural tumors (extramedullary or intramedullary), and were referred to at the beginning of this paper. This is the greatest advantage of our method, because these tumors may be early diagnosed and accurately localized at a time when all characteristic localizing physical symptoms are wanting. In such cases the stoppage is complete, and all the lipiodol gathers at the upper limit of the tumor, sometimes outlining the pole of a neurofibroma or showing a straight, transverse limit. By such means it is possible to localize the obstruction in relation to the vertebrae. From an operative standpoint this is of the greatest

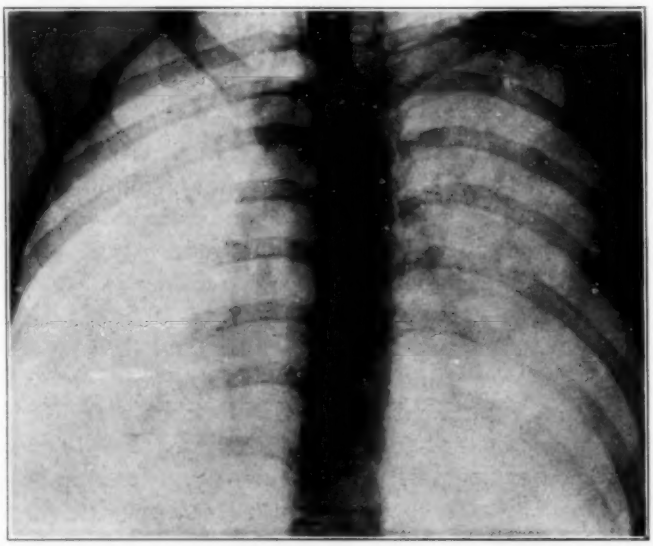


Fig. 3.—Subarachnoid space. Extramedullary tumor (neuroglioma of a thoracic root); radicular pains without paraplegia, no physical symptoms; intracisternal injection of 1.5 cc.; frontal view—complete stoppage at the level of the eighth thoracic spinal process.

interest because the exact position of the spinal segment with relation to the bones is by no means ascertained, even when the neurologic examination may enable one to determine the segmental level. Of thirty-seven patients operated on by Robineau (Paris) after lipiodol subarachnoid exploration, we had no single mistake as regards the localization of the obstruction.

Among these patients, more than half had no paraplegia, but only radicular pains with physical symptoms. One third of the whole (thirteen) had as yet no objective disturbance in sensibility; these patients could not have been operated on so early without our method, because the localization of the obstruction would have been very inaccurate by neurologic examinations.

2. Extradural compressions give subarachnoid block as revealed by lipiodol much later than intradural tumors. In Pott's disease, the recent works of Madame Sorrel-Dejerine have proved that the subarachnoid space was blocked in the course of Pott's disease paraplegia only when the affection was in evolution. In cancer of the spine with destruction of one or several vertebral bodies, the stoppage of the lipiodol happens at a rather late period in the evolution of the paraplegia.

In fractures of the spine, without dislocation, the lipiodol test may give a good account of the status of the spinal cord and a hint concerning

A B

Fig. 4.—Subarachnoid space. Intramedullary tumor (glioma); paraplegia. Intracisternal injection of 1.5 cc.; *A*, frontal view; *B*, lateral view. Characteristic picture of intramedullary tumors; operative control.

eventual operation. But the value of the test in this case is not as evident as for intradermal tumors.

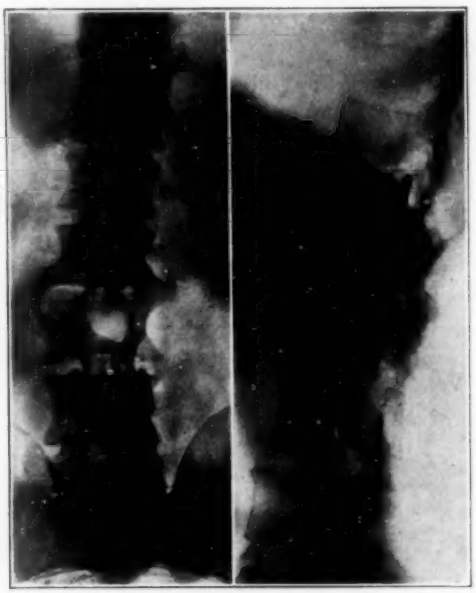
II. After Lumbar Injection: The localization of an occlusion by lumbar injection is not quite so safe, except when the operator can perform the exploration on a special radiologic table on which the patient may be put in head-down position up to an angle of 60 degrees; the sinking of the lipiodol may be easily watched by fluoroscopy. In case of pathologic stoppage, a film should be taken without changing the position

of the patient; the lower limit of the obstacle can then be accurately determined and the interpretation will be the same as before.

If the inclination of the patient has not been sufficient, the lipiodol is seen on the screen and on films, as long lines, or a series of elongated drops which can be of no significance in diagnosis.

Whenever intracisternal injection is possible without danger, it should be preferred to lumbar injection.

Our conclusion concerning the interpretation of pictures of lipiodol stoppages in spinal cord compression is that lipiodol demonstrates the compression and demarcates its limits; this is of great practical value



A B

Fig. 5.—Subarachnoid space. Fracture of the spine (war invalid); spastic paraplegia. Intracisternal injection of 1.5 cc. *A*, frontal view; *B*, lateral view. Complete stoppage demonstrating subarachnoid block from extradural compression.

to the surgeon. But it does not seem possible at present to base a positive causal diagnosis on the shape of the globule of lipiodol, this diagnosis belonging to the clinical investigator.

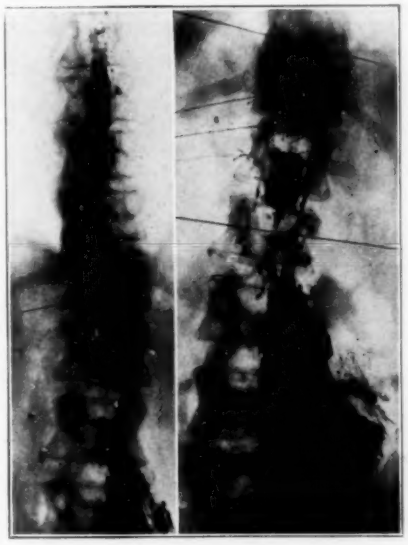
LIGHT LIPIODOL

In 1924, with L. Binet, we experimented with a dilution of lipiodol in olive oil containing 11 per cent of iodine, but having the property of floating instead of sinking in the cerebrospinal fluid. The density is a little below that of the fluid and after a lumbar injection, with the patient in a sitting position, it ascends in the vertebral canal and rises to the lateral ventricles. This solution is not so fully impervious to roentgen

rays as ordinary lipiodol; consequently, it is not easily seen in the spine, but it is satisfactorily visible in the ventricles.

When a spinal cord compression is suspected we do not advise the use of light lipiodol first. It seems safer to perform first the test with an intracisternal injection of ordinary lipiodol, and if the stoppage of the globule has demarcated the upper border of the compression, the use of light lipiodol may then help to demarcate the lower border.

The injection must be made in the lumbar segment in the patient in the sitting position. The dose to be injected is about 5 cc.; it is of no value to withdraw any quantity of fluid, but it is necessary to keep the needle and syringe in place for from three to five minutes after the



A B

Fig. 6.—Subarachnoid space. Extramedullary tumor: *A*, intracisternal injection of 1.5 cc. of ordinary lipiodol; the upper limit of the tumor is outlined; *B*, second injection, lumbar injection of 3 cc. of light lipiodol. The shape of the tumor between the two kinds of lipiodol is accurately determined; the large opaque spots at the lowest part of the pictures represent intramuscular lipiodol, previously injected for therapeutic purposes.

injection to prevent the oil from leaking out of the subarachnoid space through the hole made by the needle. The roentgenogram is taken half an hour later in a vertical position.

In normal subjects the lipiodol will be found in the lateral ventricles at the top of their cavities. A little later, we will outline the use of this method of exploration.

In pathologic obstruction, light lipiodol demarcates the lower limit of the obstacle. Combined injections of ordinary lipiodol (intracisternal injection) and light lipiodol (lumbar injection) may outline the com-

pressing tumor completely. This method is also advisable when multiple tumors are suspected; the stoppage, at long distances from one another, of the two kinds of lipiodol would draw the physician's attention to the possibility of several tumors causing compression.

ELIMINATION OF THE LIPIODOL INJECTED

We said at the beginning of this paper that lipiodol is very slowly absorbed and eliminated. Certainly, lipiodol is frowned on by some experimenters on account of the remote possibility of its acting as a "foreign body." As we have followed our patients for years, and have noted their condition and the fate of the lipiodol, we feel quite secure on this point. It is a fact that 1 or 2 cc. of lipiodol injected into the cerebrospinal fluid may remain visible for months and even for two or three years. But careful study of the urinary elimination of iodine shows that there is a constant and continuous, though very small, elimination. The daily quantity which is, in the first week, 2 or 3 mg. for 2 cc. of lipiodol, drops later to 1 mg. and even less. But it is important to note that iodine can be detected in the urine as long as lipiodol is roentgenologically visible inside the dura. This is the best proof that the oil is not, in fact, encysted.

Our anatomic and histologic investigations, with the collaboration of Laplane, have been made on either operative cases or postmortem examinations. Sometimes after from two to three months, the lipiodol is free in the fluid; sometimes it is fixed by a light arachnoid network, which contains a high percentage of mononuclear cells. In no case have we found a real cyst, or any growth such as mineral oils often cause in other tissues.

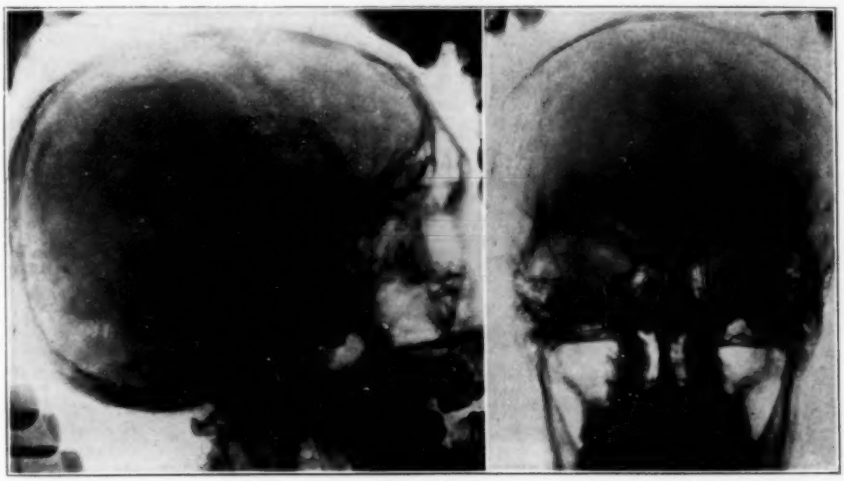
Finally, we may emphasize the fact that after three years, none of our patients showed any clinical disturbance from the presence of the lipiodol. As far as we can say, it takes about three or four years to eliminate 2 cc. of lipiodol from the subarachnoid space. When the oil remains movable for a long time the elimination may even be a little slower.

CEREBRAL VENTRICLES

When we began our experimentation with lipiodol we tried to get good pictures of cerebral ventricles through direct intraventricular injection after trephining the occipital bone. But this attempt was far from being as successful as that with the spine. The injection of from 3 to 4 cc. of lipiodol into a ventricle does not afford a good view of the cavity; all the lipiodol gathers in one part of the ventricle, and very quickly drops down to the spine through the cerebral foramina (Monro, Magendie, Luschka) being found finally in the sacrovertebral angle. In

cases of hydrocephalus, with obstruction of these foramina, the lipiodol remains in the ventricles.

We have pointed out that the dilution of lipiodol we have called "light lipiodol" when injected by lumbar puncture ascends the spine and may penetrate into the cerebral ventricles. The technic is the same as that previously described, and the dose to inject is about from 4 to 6 cc. In normal cases, both ventricles show part of the lipiodol, rather equally divided; on lateral views, the lipiodol gives the picture of a half-moon at the top of the ventricles. With pictures taken in different positions of the head, it is possible to secure inaccurate information about the shape of the ventricles. The reaction after the injection is somewhat greater



A B

Fig. 7.—Cerebral ventricles. Normal. Lumbar injection of 5 cc. of light lipiodol, emulsioned with the patient's cerebrospinal fluid. *A*, lateral; *B*, frontal view. The shape of the ventricles is accurately shown.

than with ordinary lipiodol; the rise in temperature may be 2 degrees, and a severe headache for two days is the rule, though it does not appear to be dangerous. We have not been able with this method to localize a cerebral tumor.

During the last months, with Haguenan, we have made new attempts, using an emulsion of lipiodol in the patient's cerebrospinal fluid. The technic we have followed is: After lumbar puncture in a sedentary position, 5 cc. of fluid is withdrawn, the needle being kept in place and obturated. The fluid is mixed with 5 cc. of light lipiodol in a sterilized capsule or in a 20 cc. syringe, and the emulsion which results from this mixture is reinjected immediately by means of the same needle, with the same cautions as given already for the injection of "light lipiodol."

With this method the shapes of both ventricles are perfectly outlined within two hours and the results are about the same as those of ventriculography with Dandy's method.

It is yet too soon to form a definite opinion as to this new technic. It seems to be equal in results to the injection of air. But we must say that the danger is greater than with the ordinary use of lipiodol. The reaction as to temperature and headache is generally severe; in one case of cerebral tumor a fatal result occurred within four days. The necropsy did not prove that the injection of lipiodol was the cause of the death, but we had the impression that it had been ill tolerated.

This was the only exception to a harmless result we ever had in our experimentation. Ordinary lipiodol is quite harmless; light lipiodol, especially when used in an emulsion, may give dangerous reactions.

As a conclusion, we may say that for ventricular exploration the injection of lipiodol has not given such good results as the injection of air.[4] The contrary is true for the spinal spaces.

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4. The following references to papers on the different applications of lipiodol will be of interest:

General Method.—Sicard, J. A., and Forestier, Jacques: General Method of Radiologic Exploration with Iodized Oil, Bull. et mém. Soc. méd. d. hôp. de Paris **46**:463 (March 10) 1922. Forestier, Jacques: Iodized Oil in Radiologic Practice, Paris méd. **51**:377 (April 26) 1924. Sicard, J. A.; Fabre, and Forestier, Jacques: Lipodieresis in Man, Bull. Soc. de chim. biol. de Paris **5**:413 (May 12) 1923.

Epidural Space.—Forestier, Jacques: The Intervertebral Foramen, Thesis, Faculty of Paris (Jouve, editor), 1922. Sicard, J. A., and Forestier, Jacques: Radiologic Exploration with Iodized Oil, Presse méd. **31**:493 (June 2) 1923.

Subarachnoid Space.—Sicard, J. A.; Forestier, Jacques, and Laplane, J.: The Localization of Spinal Cord Compressions with Lipiodol, Neurological Meeting of Paris, June 9, 1923. Sicard, J. A.; Paraf, J., and Laplane, L.: A Few Technical Points in the Exploration of the Subarachnoid Space with Lipiodol, Presse méd. **32**:885 (Oct. 24) 1923. Laplane, L.: Radiodiagnosis of Intraspinal Affections with Subarachnoid Injection of Lipiodol, Thesis, Faculty of Paris (Legrand, editor), 1924. Forestier, Jacques: What Becomes of Lipiodol in the Cerebrospinal Fluid, Progrès méd. **22**:802 (May 30) 1925. Dandy, W. E.: The Diagnosis and Localization of Spinal Cord Tumors, Ann. Surg. **81**:223 (Jan.) 1925. Mixter, W. J.: The Use of Lipiodol in Tumor of the Spinal Cord, Arch. Neurol. & Psychiat. **14**:35-45 (July) 1925; Compression of the Spinal Cord by Tumor, Ann. Surg. **80**:865 (Dec.) 1925.

A SIGN AND SYMPTOM OF SPINAL CORD TUMORS *

WALTER E. DANDY, M.D.
BALTIMORE

A recent case of spinal cord tumor called to my attention a subjective story and objective findings which would appear to be significant in the differential diagnosis of spinal cord tumors: sudden signs and symptoms of spinal cord compression after straining. These disturbances are emphasized because they are such logical expectations in the light of certain recent observations which have been steadily clearing the mysteries in the diagnosis and localization of spinal cord tumors.

REPORT OF CASE

Clinical History.—The patient, a well nourished man, aged 27, was referred to me by Dr. C. M. Byrnes because of inability to walk, of difficulty in urination and of a heavy dragging sensation in his back. Seven years before when straining at stool he suddenly lost the use of both legs; the arms were unaffected. On awaking the next morning the function had partially returned. At the end of four months his paralysis had improved to such an extent that he could play tennis without handicap. But during the early months of recovery there were several spells—always at stool—in which there was partial loss of function of the limbs, with gradual return to the previous normal during the course of two or three days. The motor weakness was always somewhat greater on the left side. Numbness of the limbs at times accompanied the motor loss, but the patient was never able to recall any occasion when a sensory level existed. At the onset of the paralysis and for several weeks thereafter, he suffered greatly from pain in the lumbar region, but none elsewhere. There was also some difficulty in starting the flow of urine, but no incontinence.

For more than six years after this illness the patient was free of symptoms except at times an urge to urinate and a difficulty in starting the flow. Four months ago, when at stool, partial paralysis of the legs again suddenly appeared and again the weakness of the left leg was a little greater. Numbness and tingling were also present, more on the left. There has since been but little return of function, although many times at stool the paralysis has suddenly increased; however, in twenty-four or forty-eight hours the previous level of function has nearly been restored. Vesical and rectal control has not changed.

Examination.—The neurologic examination by Dr. Byrnes revealed: marked weakness and spasticity of both legs, greater on the left; loss of muscle sense in the toes of the left foot, this was preserved on the right; positive Romberg sign; bilateral ankle clonus and increased patellar reflexes. A vague sensory level was determined about the umbilicus—tenth thoracic segment.

The spinal fluid was clear with four cells, globulin positive and Wassermann test negative.

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* From the Johns Hopkins Hospital and University.

Queckenstedt's Test: There was no rise of spinal pressure when both jugular veins were compressed.

Spinal Air Test: Thirty cubic centimeters of fluid could be aspirated; no more was obtainable. An equal amount of air was substituted in the spinal canal. Only after several minutes was there a slight headache. But since air could be repeatedly reaspirated from the spinal canal it was evident that it was held below a block in the spinal canal. Roentgenograms of the head disclosed only a small amount of air in the cerebral sulci. We were, therefore, dealing with a spinal block which was nearly but not quite total.

Extradural spinal cyst, on which the reported hydrodynamic observations are largely based; the cyst is apparently of congenital origin; it is attached at only one point—the sheath of a spinal nerve; it blocks the spinal subarachnoid space though the obstruction is not absolutely complete.

Operation.—A simple cyst, measuring about 6 by 2 cm., of oval shape, with a rather thick but slightly translucent wall and containing a clear slightly straw colored fluid, was dissected without rupture from the epidural space at the seventh and eighth vertebrae. It was only loosely attached to the dura and could be easily lifted from its bed. It was, however, attached to the sheath of the left seventh thoracic nerve and here alone was sharp dissection necessary for liberation of the tumor. In neither the gross nor the microscopic examinations could any solid element of tumor be found. Microscopic sections of the wall show only a hyaline fibrous tissue, and no nerve elements.

Anatomic Diagnosis.—Simple cyst—extradural. It would seem that this tumor was probably of congenital origin.

Result.—The patient walked out of the hospital two weeks later and now (after one year) has completely recovered all motor functions and is well in every respect.

COMMENT

The unusual character of this tumor need not detain us, nor shall I dwell either on the unusual history of a series of sudden attacks quickly followed by recovery instead of the characteristic tumor history with signs and symptoms of gradual progression, or on the equally unusual fact that there was a long free interval of seven years between attacks of paraplegia. The character of the tumor—a simple cyst—well explains these seemingly nonharmonizing facts.

We are here concerned solely with the sudden disturbances of spinal cord function associated with straining as a diagnostic sign of spinal cord tumor. In this case the paraplegia occurred so frequently and so consistently when at stool that coincidence cannot be offered as an explanation. The explanation of these changes has been prepared in recent years by the hydrostatic and hydrodynamic studies of Queckenstedt [1] and of Ayer.[2] As a matter of fact precisely the same loss of spinal cord function has quickly followed lumbar punctures in the presence of spinal cord tumors and for precisely similar reasons. This startling and not unusual sequel of a seemingly simple diagnostic procedure has been emphasized elsewhere (Elsberg,[3] Dandy [4]).

From the frequent use of Queckenstedt's test we know that compression of both jugular veins produces: (1) an instant and rapid rise of fluid in the spinal manometer when the spinal subarachnoid space is not blocked; (2) but no change in the column of fluid if the spinal canal is blocked by a tumor or other lesion. Since the jugular veins have no valves, pressure on them causes blood to be held back within the cranial chamber (a closed and inexpansible box), and therefore, the intracranial pressure must at once rise. And because of the normal free communication between the cerebral and spinal subarachnoid space,

1. Queckenstedt: Zur Diagnose der Rückenmarkskompression, Deutsche Ztschr. f. Nervenh. **55**:325, 1916; über Veränderungen der Spinalflüssigkeit bei Erkrankungen peripherer Nerven, insbesondere bei Polyneuritis und bei Ischias, ibid. **57**:316, 1917.

2. Ayer, J. B.: Puncture of the Cisterna Magna, Arch. Neurol. & Psychiat. **4**:529 (Nov.) 1920; Spinal Subarachnoid Block as Determined by Combined Cistern and Lumbar Puncture with Special Reference to the Early Diagnosis of Cord Tumors, Arch. Neurol. & Psychiat. **7**:38 (Jan.) 1922.

3. Elsberg, C. A., and Stookey, B.: Mechanical Effects of Tumors of the Spinal Cord; Their Influence on Symptomatology and Diagnosis, Arch. Neurol. & Psychiat. **8**:502 (Nov.) 1922.

4. Dandy, W. E.: The Diagnosis and Localization of Spinal Cord Tumors, Ann. Surg. **81**:223 (Jan.) 1925.

the intracranial pressure must be transmitted at once to the lumbar spinal canal.

When a spinal puncture is made in the presence of a spinal tumor, the pressure below the tumor is reduced to that of the atmosphere; the pressure above the tumor, always higher, then becomes relatively greater and the full force of this difference is suddenly exerted on the point of obstruction. The result of this pressure is trauma to the spinal cord whose functions are already greatly compromised by the tumor's gradual pressure. This injury is the more readily explainable when one considers the anatomic changes in the spinal cord at the site of the tumor. The spinal cord is always deeply indented by the gradual growth of the tumor to which it must, of course, make an accurate physical adaptation. This involves a marked loss in volume of the spinal cord, though function may still be retained. At the level of the tumor's maximum and the spinal cord's minimum diameter is, of course, the narrowest part of the spinal canal, and through this hour-glass constriction the pressure, now suddenly relatively increased, must try to jam the greater volume of spinal cord as a wedge. The result of this hydrodynamic effort to equalize pressures above and below the tumor is trauma to the delicate spinal cord. The reason an injury of this kind does not always follow is doubtless because the spinal canal may be hermetically closed at the level of the tumor so that the newly established pressure differential can have no effect, or the canal may be still sufficiently patent to permit the ready transfer of fluid, thus equalizing the pressure above and below the tumor.

In our patient a similar pressure differential has been created, but in a different manner. A necessary sequel of straining at stool is increased intracranial pressure from thoracic compression (venous congestion)—the exact equivalent of compressing the jugular veins. Nature has then raised the spinal pressure above the tumor (that below being unaffected). The end-result has, of course, been the same—the spinal cord is driven by the increased pressure into the constriction of the spinal canal at the site of the tumor, and, just as following a lumbar puncture, the spinal cord is traumatized.

In a recent survey of a series of spinal cord tumors no instance of this effect of straining at stool was observed, nor has its mention been noted in a review of the literature on spinal cord tumors.

One of my patients volunteered the statement that frequently when he sneezed or coughed violently, pains and numbness immediately developed in both legs but quickly disappeared. This history was given some time before the other patient's story was known and at the time was passed by as of no significance. The intracranial pressure is, of course, increased by coughing and sneezing in just the same way as by straining

at stool and doubtless the pains and numbness of our second patient are explainable in precisely the same way as in our first case. The order in which the signs and symptoms of spinal cord compression develop must, of course, be variable and doubtless dependent on the position of the tumor; i. e., whether dorsal, ventral or lateral. It would, therefore, seem reasonable to expect motor symptoms to be initiated by a ventral tumor, Brown-Sequard symptoms by a lateral tumor and possibly sensory symptoms by a dorsal tumor.

At times a localized backache is intensified by coughing, sneezing or straining at stool when the patient is affected with a spinal cord tumor. This was mentioned in the history of our patient. Schultze [5] and Cassirer [6] mention cases in which root pains are intensified by coughing and sneezing. Backache in spinal cord tumors appears to be due to localized intraspinous pressure. An intensification of this pain would, therefore, be expected by the sudden increase of intraspinous pressure above the tumor after sneezing, coughing or straining at stool. Edema of the cord, resulting from the trauma, causes an increased swelling of this local area of the spinal cord and, therefore, a persistence of the backache for sometime thereafter. All forms of pain, which are caused by spinal cord tumors—(1) root pains, (2) tract pains and (3) the pressure pains (backache)—may be intensified by coughing, sneezing and straining at stool. The explanation of the increased pain is precisely that of the sensory, motor and vesical disturbances, i. e., the effect of increased pressure above the tumor.

It is true that the repeated attacks which occurred in our patient will never develop in the usual run of tumors whose growth is progressive, but I am prepared to believe that a history of at least one and possibly even more attacks, of more or less similar character, may at times be elicited if this symptom is suspected by the examiner. And such a story will, I believe, be of great importance in differentiating a spinal cord tumor from other spinal lesions.

As mentioned before, when the spinal canal is tightly closed or when it is yet patent, it is improbable that injury to the spinal cord can result either by lumbar punctures or by straining. In other words the stage, at the tumor's level, must be set to a nicety before the changed intraspinal pressure differential can produce these disturbances of spinal cord functions. The intensified backache, however, should be expected at any time after the original cause of the backache exists; i. e., after the com-

5. Schultze: Zur Diagnostik und operativen Behandlung der Rückenmarkshautgeschwülste, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **12**:153, 1903.

6. Cassirer: Die Tumoren des Rückenmarks, Oppenheim's Lehrbuch der Nervenkrankheiten, ed. 7 **1**:521, 1923.

bined volume of tumor and cord is great enough to cause marked intraspinous pressure which can no longer be relieved by nature's method of space compensation.

THE EFFECT OF STRAINING IN THE PRESENCE OF INTRACRANIAL TUMORS

All that has been said concerning the effects of increased intracranial pressure on the spinal cord can be transferred almost verbatim to apply to tumors of the brain, particularly those situated in the posterior cranial fossa. The not infrequent disastrous effects of lumbar punctures on patients with intracranial tumors are too well known to need mention. The deaths and disabilities are likewise explained by trauma to the medulla when the cerebellar tonsils are jammed into the bony ring forming the foramen magnum. The effect is precisely that of injury to the spinal cord following lumbar puncture in the presence of a spinal cord tumor. The result is more serious because the intracranial pressure is much higher, but particularly because injury to the medulla compromises life.

But the increased intracranial pressure caused by straining produces equally disastrous results which have an explanation similar to those following lumbar punctures. During one year, two of our patients suddenly died on the morning of operation when at stool; another passed into coma from which she was rescued by operation. In each of these patients the tumor was of long standing; nor had there been any apparent change in the patient's condition prior to the time of defecation. The sudden, even though transient, increase of pressure was enough to endanger or terminate life—"the straw that breaks the camel's back."

That there may even be a local intracranial pressure effect from straining is shown in a recent patient with a cerebellopontile tumor. For years she dreaded going to stool because a headache always developed and persisted for several hours in the right suboccipital region (the site of the tumor).

SUMMARY AND CONCLUSIONS

1. In a case of tumor involving the spinal cord, signs and symptoms of spinal cord compression repeatedly and suddenly developed when the patient strained at stool. In another case numbness and pain developed in both legs immediately after sneezing.

2. These signs and symptoms are explained by a sudden increase of the intraspinous pressure above the tumor, and caused by straining at stool, coughing or sneezing.

3. This increased pressure forces the spinal cord downward against the spinal tumor causing trauma of which the disturbed function—motor, sensory and sphincteric—is the result.

4. For similar reasons, root pains, tract pains and backache—the three pains of spinal cord tumors—may follow sneezing, coughing and straining at stool.

5. It seems probable that the sudden onset of any of these disturbances from straining may be a sign of importance in differentiating tumors from other lesions affecting the spinal cord.

A SATISFACTORY METHOD FOR STAINING THE NERVE FIBERS OF THE IRIS *

MANUEL BALADO, M.D.
Special Student in Experimental Surgery, the Mayo Foundation
ROCHESTER, MINN.

The nerves of the iris, discovered by Petit [1] and described by de Saint-Yves,[2] have been the object of a great number of discussions, in regard to their existence, course and relationship with the other parts of the iris. The reason for these discussions is the lack of a staining method that gives consistent and uniform results and that is applicable to the pigmented iris of mammals. A review of the literature on this subject shows that our knowledge of the nerves of the iris is dependent on descriptions of the iris of albino rabbits and mice, or on incomplete studies on the pigmented iris. Furthermore, the results obtained by several investigators on the study of the naturally depigmented iris, depend on unreliable procedures.

In 1877, Pause [3] employed a defective method, but one which allowed him to make a sufficiently accurate description of the more important nerve plexuses of the iris. This procedure consisted in staining the nerve fibers with osmic acid. If the iris was pigmented he removed as much of the pigment as possible by stroking the posterior surface with a brush. In 1878, Formad [4] attempted to solve the problem of the pigmented iris by bleaching with chlorine, and, after treatment with acetic and chromic acid, staining with carmine and hematoxylin. This method produced good results only with the iris of albino rabbits. In 1880, Meyer [5] described the nerve fibers of the iris, and classed them in three categories: fibers that go to the sphincter muscle of the iris (motor fibers); fibers that go to the anterior surface of the iris (sensory fibers), and fibers accompanying the vessels (vascular fibers). His work is based on results obtained by the inconstant gold chloride method and is applicable only to the albino rabbit.

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* Work done in the Division of Experimental Surgery and Pathology of the Mayo Foundation.

1. Petit: Mem. Acad. roy. de sc. Paris, 1727.

2. Saint-Yves, Charles de: A New Treatise of Diseases of the Eyes, Containing Some New Discoveries in the Structure of the Eye (translated from the French by J. Stockton), London, T. Osborne, 1744.

3. Pause, C. H.: Ueber die Nerven der Iris, Berlin, H. Peters, 1877.

4. Formad, H. F.: The Distribution of Nerves in the Iris, Am. J. M. Sc. **75**: 93-100, 1878.

5. Meyer, Andreas: Die Nervenendigungen in der Iris, Arch. f. mikr. Anat. **17**:324-334, 1880.

In 1883, Gruenhagen [6] employed the same method for the ciliary process, and to stain the nerves of the pigmented iris stroked the posterior surface of the iris with a brush. In 1884, Geberg [7] employed a method very similar to that of Pause.

In 1891, Hosch [8] utilized Ehrlich's vital staining method with methylene blue. This method can only be used (with all its inconstancy) with the iris of the albino rabbits. The results were coordinate with those of the authors already referred to.

In the following year, Stepanow [9] repeated this procedure with the iris of the albino mouse. In 1897, Andogsky [10] and Agababow [11] utilized Ehrlich's method with the iris of albino animals. The first of these authors denied the existence of ganglion cells in the iris, as did Formad.

No further investigation of the nerves of the iris was made until 1911. In this year, Seligmann [12] proposed the use of the method of Golgi as modified by Cajal.

Meantime, advances in histologic technic were being made, and three procedures important in relation to staining the nerves of the iris were elaborated: (1) the procedure of Alfieri [13] for the depigmentation of the sections of the iris, (2) the technic of frozen sections and (3) the method of Spielmeyer [14] for the staining of the myelin in frozen sections. With these three procedures I have been able to elaborate a method of demonstrating the nerves of the iris that fulfils the two conditions imposed at the outset. The method, which is a slight modification of one I presented in a previous communication,[15] is as follows:

The eyeball is fixed in a 10 per cent solution of liquor formaldehydi for twenty-four hours. The eyeball is cut transversely into two parts, anterior and posterior. The choroid is detached from the sclera to the

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6. Gruenhagen, A.: Die Nerven der Ciliarfortsätze des Kaninchens, Arch. f. mikr. Anat. **22**:369-373, 1883.

7. Geberg, A.: Ueber der Nerven der Iris und des Ciliarkörpers bei Vögeln, Internat. Monatschr. f. Anat. u. Histol. **1**:7-52, 1884.

8. Hosch, F.: Ehrlich's Methylen-Blaumethode und ihre Anwendung auf das Auge, Arch. f. Ophth. **37**:37-54, 1891.

9. Stepanow, quoted by Hosch.

10. Andogsky, N.: Zur Frage über die Ganglienzellen der Iris, Arch. f. Augenh. **34**:86-98, 1896-1897.

11. Agababow, A.: Ueber die Nervendigungen im Corpus ciliare bei den Saugetieren, Internat. Monatschr. f. Anat. u. Physiol. **14**:53-70, 1897.

12. Seligmann, Sieghert: Die mikroskopischen Untersuchungs Methoden des Auges, Berlin, Karger, 1911.

13. Alfieri, quoted by Seligmann.

14. Spielmeyer, Walther: Technik der mikroskopischen Untersuchungen des Nervensystems, Berlin, Springer, 1914.

15. Balado, Manuel: Topografía de los núcleos de origen de los nervios craneanos del perro, Bol. d. inst. de clín. quir. **1**:211-227, 1925.

point of attachment of the ciliary muscle. All the choroid and the ring of the ciliary muscle are carefully detached from the sclera. As the choroid and iris have been previously fixed they conserve their shape perfectly. This procedure was described by Czermak [16] in 1885.

The iris is washed in water for twenty-four hours, then placed in a 10 per cent solution of gelatin for twenty-four hours, and then a 30 per cent solution of gelatin for twenty-four hours. Subsequently, the iris is placed in a little pasteboard box filled with a 30 per cent solution of gelatin, and is allowed to harden in the icebox. When the gelatin has hardened, the box is detached, and the embedded iris placed in a 10 per cent solution of liquor formaldehydi for twenty-four hours.

The frozen sections are made in the usual manner. The sections must be parallel to the anterior surface of the iris, because in that manner it is possible to obtain all the thickness of the iris in a very few sections. The sections are fastened to the slides by allowing them to dry in position, and then at a particular time, before the drying has gone too far, they are passed rapidly, with a glass rod, through celloidin (syrup), the surface of the sections being covered completely. The sections are then placed in a 70 per cent solution of alcohol for fifteen minutes.

The modifications of the original Alfieri method are very numerous (Seligmann). I placed the section in potassium permanganate solution, 1 : 500, for twenty-four hours, then rapidly washed it in water, and passed it into a 1 per cent solution of oxalic acid. I repeated these operations as many times as was necessary to make the sections appear white. The time that the sections must remain in the oxalic acid depends on the previous action of the permanganate. After twenty-four hours in this agent, twenty minutes in oxalic acid is sufficient. If the sections remain yellow or brown, all the depigmenting operations must be repeated.

When the sections are white, they are passed into a concentrated solution of potassium alum (10 per cent), in which they remain until they appear transparent.

The sections are placed in a solution of 2.5 per cent ferric ammonium sulphate (chemically pure, violet crystals) for two hours, then rapidly washed in water and passed into the hematoxylin of Heidenhain in which they remain twenty-four hours. After this the sections are again placed in the solution of ferric ammonium sulphate for the differentiation of the medullated nerve fibers from the other parts of the iris. When the differentiation is completed, the sections are thoroughly washed in water, then in a 70 per cent solution of alcohol, a 95 per cent solution of alcohol, carbol xylol, xylol and Canada balsam.

16. Czermak, W.: Zur Zonulafrage, Arch. f. Ophth. **31**:79-138, 1885.

THE MEDULLATED NERVE FIBERS OF THE DOG

The aspect, distribution and anastomosis of these fibers, after a reconstruction through serial sections, is that represented in figures 1 and 2.

In figure 1 the main nerve fibers of the iris are semischematically shown where they enter the iris; they number six or eight. They divide,

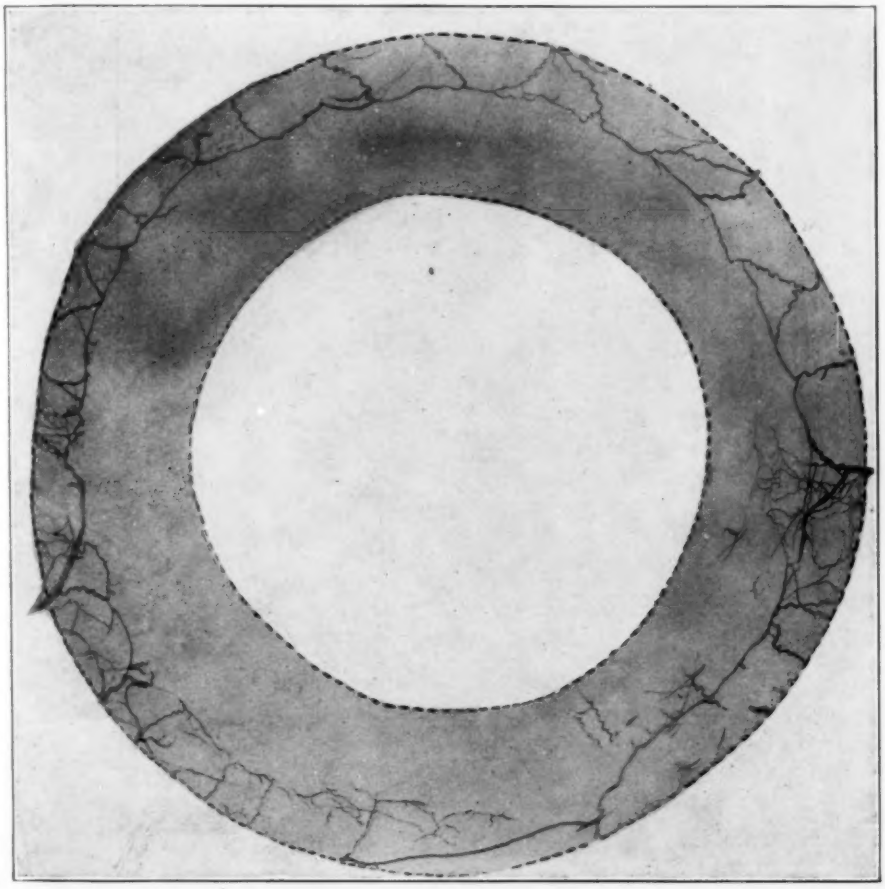

Fig. 1.—Semischematic aspect of the main trunks of the medullary nerves of the iris of the dog.

and anastomose, forming a series of arches; between these arches there are other smaller and slender tracts that anastomose between themselves and with the main branches.

In figure 2 (after reconstruction of serial sections) are shown the great and medium-sized arches, sending, concentrically to the pupillary

margin of the iris, branches of very different sizes. These branches anastomose capriciously, forming plexuses of fine fibers, especially on the most anterior part of the iris. Near the sphincter muscle of the iris, they decrease considerably in number so that little but the large

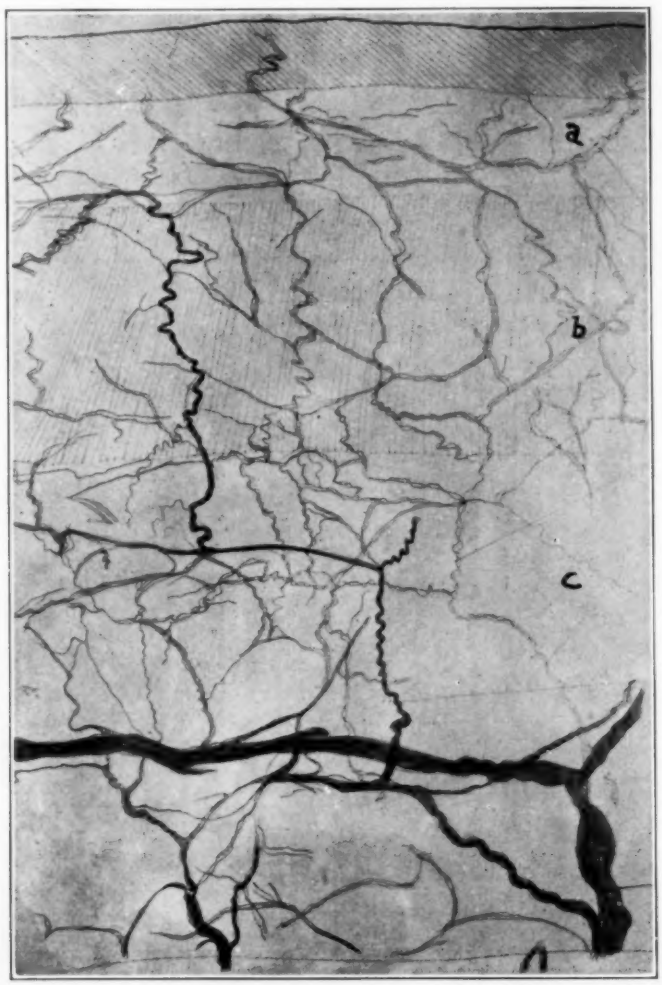

Fig. 2.—Reconstruction from serial sections of the iris of the dog, showing the medullated fibers; *a*, pupillary margin; *b*, sphincter; *c*, sclerocorneal margin.

trunks remain. They form finally near the pupillary edge a new and last plexus of medullated nerves.

In figure 2, are represented in a single plane all the medullated nerve fibers of the iris. The sinuous aspect of the fibers is due to the dilatation of the pupil in this case.

Clinical and Occasional Notes

MULTIPLE PRIMARY CEREBRAL NEOPLASMS REPORT OF A CASE*

IRVING J. SANDS, M.D., BROOKLYN
Associate in Neurology, Columbia University; Attending Neurologist, Jewish, Brownsville and United States Veterans' Hospitals

Multiple primary malignant tumors have great interest for both clinicians and pathologists. In an excellent summary of this subject Major[1] calls attention to the fact that the literature contains many such reports. He adds a case of carcinoma of the face and a round cell sarcoma of the stomach. Seecof[2] reports a case of carcinoma of the lung and carcinoma of the rectum, each producing metastases. It is not at all uncommon to find metastases in the brain, but primary multiple cerebral neoplasms are rare, though a few cases have been previously reported. Thus, Behrendsen[3] reports a case of a cholesteatoma of the pia mater in the region of the pons, and a glioma of the left hemisphere; Bartels[4] reports two cases, one of mulitple primary sarcoma of the meninges with invasion of the brain, and another of multiple endothelioma and multiple neurofibroma of the base; and Stromeyer[5] reports a case of sarcoma and cholesteatoma. Because of the infrequency of primary multiple cerebral neoplasms, and because it illustrates the importance of making a complete postmortem examination at every available opportunity, the report of the following case is presented.

REPORT OF CASE

F. W., aged 43, white, married, native of the United States, was admitted to the Jewish Hospital, Feb. 27, 1924, and died on the same date. The family and personal history was unimportant. The patient had been in excellent health until about seven weeks before admission when she had a severe epistaxis. She remained in bed for two weeks and then resumed her household duties, but complained of severe headache. She was unable to sleep at night because the headache was constant and was not relieved by medication or by counter irritation. At 9 p. m., Feb. 26, 1924, she had a convulsion in which the whole body became rigid, the face became blue, and she foamed at the mouth and was incontinent. The attack lasted a few minutes and then she regained consciousness. At 3 a. m., February 27, she had a second attack and did not

* From the Pathological Laboratory of the Brooklyn Jewish Hospital.

1. Major, R. H.: Multiple Primary Malignant Tumors, Bull. Johns Hopkins Hosp. **29**:233 (Oct.) 1918.

2. Seecof, D. P.: Multiple Primary Malignant Neoplasms, J. Cancer Research **8**:213 (July) 1924.

3. Von Behrendsen: Ein Fall von gleichzeitigem Auftreten zweier verschiedenartigen Hirntumoren, Deutsche med. Wchnschr. **25**:710, 1899.

4. Bartels, J.: Ztschr. f. Heilk. Path. Anat. **7**:281, 1905.

5. Stromeyer, F.: Über ein mit Sarkom combiniertes Cholesteatoma des Gehirnes, Beitr. z. path. Anat. u. z. allg. Pathol. **47**:391, 1910.

regain consciousness. Since the second attack she had had convulsions every half hour or so, each lasting several minutes. She was admitted to the hospital at 7:45 a. m. the same day.

Physical Examination.—The patient was well developed, well nourished and in deep coma. The respirations were regular but noisy and the skin was cyanotic; there were no cervical rigidity, Kernig sign or pathologic reflexes. The biceps, triceps and knee and ankle jerks were present and of normal type. The heart was not enlarged; râles were present in the chest. The liver and spleen were not palpable. The abdomen was soft. The pulse rate was 76, respirations 20 and temperature 99.6 F. The patient had convulsions every twenty minutes or so in which the head turned to the left and the body became rigid, followed in a few seconds by generalized clonic convulsions. There was frothing at the mouth, and incontinence of urine and feces. The convulsions lasted from thirty seconds to a minute. The pupils were dilated. A

Fig. 1.—Sagittal section of right frontal lobe showing the two different types of tumors.

second clinical note stated that the convulsions were characterized by tonic contractions, first of the arms, later of the lower extremities; this was immediately followed by clonic convulsions of all extremities. The eyeballs rolled upward and inward, and the patient became markedly cyanotic. The eyegrounds showed haziness of the disks but no real choking. Between the attacks the pupils were equal and regular and reacted well. Lumbar puncture yielded 10 cc. of clear fluid under three plus pressure. The fluid contained 30 lymphocytes, and tests revealed no globulin, a 3 mm. albumin ring, reduction of Fehling's solution and no reduction of the colloidal gold solution. The blood and spinal fluid Wassermann reactions were later reported as negative. The urine was negative and the blood count normal. At 12 o'clock the temperature had risen to 100.6 F., with pulse 140 and respirations 40. At 4 o'clock the temperature was 104 F., pulse 130 and respiration 40. The patient died in a convulsion at 4:45 p. m., February 27.

Necropsy Findings.—In addition to the pathologic changes to be described the following were found: chronic fibroid phthisis; fibrosis uteri; congestion of liver, spleen and kidneys.

The calvarium was removed with ease. The dura appeared thin and glistening, but was adherent to the right frontal pole. The brain was of normal size and weighed 1,325 Gm.; the sulci were of normal depth, and the convolutions appeared normal. The right frontal lobe felt much denser than the left. On sagittal section through the adherent dura, two tumors presented themselves (fig. 1). The tumor to which the dura was adherent was in the superior frontal gyrus, and measured 3 by 4 cm. The second tumor was situated 3 cm. below the first, and was 2 by 3 cm. in size; it was about 0.2 cm.

Fig. 2.—Low power microscopic appearance of the endothelioma.

from the orbital surface of the brain. The first tumor on microscopic examination proved to be a typical endothelioma (fig. 2). The second proved to be a typical angioma (fig. 3).

Each of these types of tumor is now well known. The endotheliomas received special study by Cushing[6] who called attention to the fact that, though attached to the dura, they do not arise from this membrane but from elements of the arachnoid which project into it. These tumors eventually burrow into the

6. Cushing, Harvey: The Meningiomas (Dural Endotheliomas), Brain **45**:282 (Oct.) 1922.

brain and may become detached from their dural pedicles. As pointed out by Goldstein,[7] angiomas of the brain may appear in different types. Oppenheim[8] called attention to the slow, symptomless course that angiomas may run; they may fail to produce the so-called general symptoms of brain tumor. Only the occurrence of hemorrhage or thrombosis may elicit symptoms; there are often jacksonian seizures, hemianopia and hemiparesis.

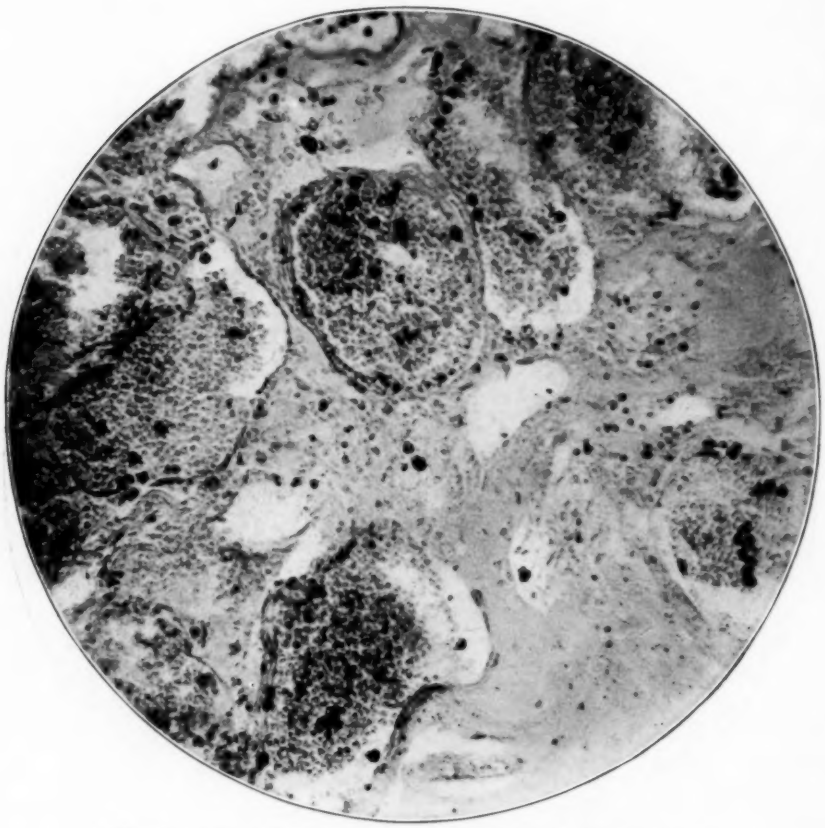

Fig. 3.—Low power microscopic picture of the angioma.

SUMMARY

Multiple primary cerebral neoplasms are relatively rare. A case is reported showing an endothelioma and an angioma in one frontal lobe producing at first severe nose bleed, followed by headaches, extending over a period of seven weeks, and finally by repeated convulsions and unconsciousness for eighteen hours, resulting in death.

202 New York Avenue.

7. Goldstein: Lehrbuch der Nervenkrankheiten, ed. 7 (German) **2**:1384, 1923.

8. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, ed. 7 (German) **2**:1449.

SPECIAL ARTICLE

NEUROPATHOLOGY—II *

SAMUEL T. ORTON, A.M., M.D.
IOWA CITY

As suggested in the previous article the phylogenetic translation of the centers for various sensory functions from the lower levels (midbrain nuclei) to the cerebral mantle does not occur at one time. The first true forebrain structures to be developed in the animal series are devoted to the function of olfaction, and apparently this cortex is another illustration that certain of nature's structural experiments in the lower animals are incorporated in the higher without great modification. Thus the cortices serving olfaction in the mammalia are characterized by a very different cyto-architectonic design from that of the rest of the cerebral mantle. To this phylogenetically old cortex is given the descriptive name of the archipallium. All the rest of the mantle is formed by minor modifications of a single more recent pattern and is known as the neopallium. Figure 1, which is taken from Edinger, shows the comparative extent of the archipallium and the neopallium in four steps of the phylum.

With the translation to the cortex of the controlling centers for all sensory functions there arises the very complex and intimately related group of physiologic centers which constitutes the mammalian brain. The study of the distribution and functions of these areas constitutes one of the most intricate and entrancing fields of biology—cerebral localization. No single method of approach has proved adequate for this study and our views of today are derived from the comparison of data garnered in several fields of observation. Five of these, the method of anatomicoclinical correlation, the experimental, the anatomic, the myelogenetic and the comparative anatomic have been most prolific, and it may be well to look into each method somewhat critically.

I. Anatomicoclinical Correlation.

This method as its name implies consists in the comparison of symptoms observed during the illness of a patient with lesions found in the brain at necropsy. The elements of error which enter here are quite large and may be presented under two heads, the clinical and the anatomic.

* Second installment of a series on this subject from the State Psychopathic Hospital, State University of Iowa. The first article appeared in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, June, 1926.

Our clinical methods must, I think, be recognized as very coarse when compared to the intricacy and refinement of the functions we are attempting to study and there is evidence, in the majority of case reports, of a lack of thoroughness on the part of the examiner whose attention has been caught by one phase of the disease process and who, while recording this faithfully, has failed to make note of the adequacy of certain other related functions. Head has called attention to this in

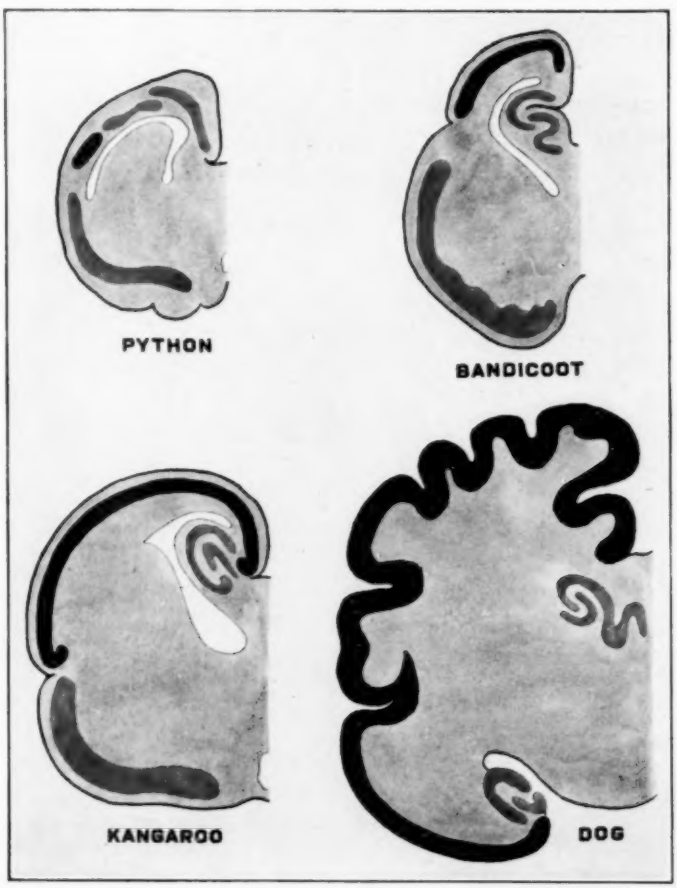

Fig. 1.—Comparative development of archipallium (black) and neopallium (dark gray) in a reptile, two marsupials and a mammal (after Edinger).

his work on aphasia and has published a series of tests for aphasic patients which he recommends as a protocol for records in such cases.

On the anatomic side there are also many sources of error. Relatively few lesions of the brain are pure in the sense of being sharply restricted to areas of one function. The vascular lesions which form so large a part of our case studies are practically never so. Tumors,

gummas, tubercles and abscesses are second, while traumatic cases, particularly certain war wounds, form the purest series. All types are prone to involve not only one cortical locus but also paths of communication or association between other cortices which lie beneath that locus. In one recorded case, for example, a through and through gun-shot wound which destroyed part of the angular gyrus of both hemispheres, the published interpretation is based largely on the effects of cortical destruction with little analytic attention to the 6 inches (15.2 cm.) or more of fiber paths which must have been cut between these areas.

The lesions due to vascular obliteration are rarely confined to areas of one function. This is particularly true along the Sylvian fissure where areas of different function are closely approximated. In a general way the major blood vessels feed certain general functional zones as, for example, the anterior cerebrals which supply the frontal fields to a large extent, but it is quite obvious that cortical differentiation is not greatly influenced by vascular supply, nor is the vascular branching controlled by the functional differentiation of cortical areas. Thus the plugging of a branch of the Sylvian artery which passes toward the overlying opercula rarely results in a softening of cortex of one structural type, but involves frontal, precentral, postcentral and parietal in varying degrees.

Moreover, most vascular lesions are the result of diffuse disease of the blood vessels with varying degrees of severity in different loci. There is, therefore, a great tendency to multiplicity of lesions. All too frequently the occurrence of one outstanding symptom has caught the attention of the clinician and a search is made for a lesion in an indicated area with little or no regard to the possible effects of other lesions which are found. One naive report in the literature is that of a case which exhibited a specific form of apraxia clinically and which at death was considered to uphold the reporter's thesis for the localization of this function because of the occurrence of a softening in this area although the necropsy report indicated that there were seven other areas of softening.

Another important factor of confusion in the vascular lesions is that of the frequent occurrence of fine microscopic lesions in nerve cells which cannot be determined in the gross and some of which can only be demonstrated by special technical methods and could scarcely be observed in total brain sections stained by the ordinary procedures. One type of such lesion is that found in the small, widely scattered, wedge shaped areas of cell devastation so common in many cases of arteriosclerosis and resultant from partial or complete obliteration of small branches of the arterial tree. Even more difficult to evaluate are the results of diffuse intracellular processes which give a gross picture of cerebral atrophy without local destruction but which at the same time give rise

to symptoms with distinctly localizing clinical inference. Alzheimer's disease forms an illustration of this type. Diffuse basket cell degeneration which can be demonstrated in greater or less degree in almost all cortical areas and yet without coarse focal lesions is here related with the clinical occurrence of the alpha privative symptoms—alexia, agraphia, apraxia, etc.—which are usually taken to indicate focal destruction. I know of no exhaustive study of the relative numerical severity of cell degenerations in such cases, but such a study might readily prove the cellular degenerations to appear in certain areas with sufficient concentration to explain the focal symptomatology.

Few recorded cases of the past have had adequate histologic studies to control the part played by fine lesions in the genesis of symptoms in cases which also show coarse focal lesions.

II. The Experimental.

Certain broad limitations to the field of animal experimentation are immediately obvious. Many of the most important activities of the human mind—speech, writing, reading, etc.—can, of course, not be approached from this direction. Certain others, for example, the motor functions, and the lower and less elaborated functions of the visual and auditory spheres may be studied in animals but often the results are not to be directly applied to man. For example, in volitional motion, the degree of control exhibited by the human cortex over the lower motor centers is much higher than that in the lower animals as evidenced first by the period of recovery from spinal shock and second by the amount of recovery of function after cortical ablations. I think we must recognize here the general rule indicated by the phylogeny of the forebrain, that the midbrain centers are much more competent in the lower animals and hence lesions of the forebrain will give less severe symptoms and more rapid recovery. Man's brain standing at the head of the list represents the highest structural development of the cortex and as a corrollary of this a much more exclusive control of function from this level. As a result, lesions of cortical areas in the lower mammals and even in the monkey give rise to lesser functional limitations and greater tendency to recovery than in man. The results of motor cortex ablations in the monkey or ape are a case in point.

Two chief methods of experiment have been applied to animals. The first is that of stimulation of cortical areas with observations of the responses. Electrical and chemical stimulation have both been used and are open to the criticism that there is great difficulty in so controlling the intensity of the stimulus that its influence affects only the area of its application and not, by spread of discharge, adjacent intimately related areas. Our present view of the cortex of the postrolandic lip is that it is of exclusively sensory function and yet its connections with the cor-

responding motor areas of the prerolandic zone are of great wealth, and the earlier stimulation experiments gave rise to motor reaction when the electrodes were applied behind as well as in front of this fissure. The confusing results from this spread of discharge are readily seen in all the older maps of this zone in which the motor areas are shown extending across the fissure of Rolando and occupying what we now regard as cortical areas of two distinctly differing functions.

A few stimulation experiments have been carried out in man in cases in which the brain has been exposed for other reasons but no broad program is possible here.

A second method carried out in the experimental animal is that of producing destructive lesions and observing the clinical effects. This has the great advantage that the results of such destructions can be carefully controlled by both gross and microscopic studies post mortem. Where this control has not been applied the results are not of much value. This procedure gives, of course, accurate information concerning the anatomic connections between areas but it suffers from the limitations of all work on the lower animals which have already been pointed out.

III. The Anatomic.

In one particular cortical field, the area striata or calcarine cortex, there is a striking structural feature of such prominence that this area can readily be delimited by the naked eye. This is the white line of Gennari or the stria of Vic d'Azyr and appears as a clear white line about midway in the depth of the cortex Similar but much less prominent differences can be noted by careful observation in other cortical fields and Elliott Smith has stated that a considerable degree of cortical differentiation can be attained by such naked eye examination. This method cannot, however, be said to be accurate except possibly after long training, and it has not found great favor.

Analytic studies with the microscope have carried our knowledge of structural differences between cortical areas much further and form, I think, the most convincing evidence of differences in function. The precise correspondence between the numbers, type and stratification of nerve cells and fibers from the same area in different brains and the striking differences in these same characteristics between two areas of the same brain carry the conviction that these differences and likenesses occur because of the function to which each area is devoted. The names of Meynert, Bevan Lewis, Cajal, Campbell, Brodmann, the Vogts and many others are associated with histologic localization. Bevan Lewis in 1879 stated that, "No abrupt passage from one form of cortical lamination to that of another is ever seen in the convolutions of the vault. Changes in type are gradually assumed and no distinct boundary

line exists to indicate an exact line of demarcation. The structure peculiar to one region gradually fades and blends with arrangements which foreshadow the architecture of adjacent realms and thus we obtain transition regions." This statement is no longer tenable as regards certain fields. For example, the loss of the granule layer of the postcentral zone as the area of precentral type is reached at the bottom of the fissure of Rolando is sharp and a very exact line of demarcation can be readily observed in passing from the calcarine cortex to the surrounding occipital areas. In many others, however, Lewis' statement will still hold although progressive refinement in studies of the details of cell and fiber equipment has served to point out minor differences and hence to subdivide many large fields A comparison of the maps of Campbell and Brodmann will illustrate this tendency.

The histologic method is the most accurate but it will, of course, only identify structural differences and while this implies difference of function the studies of structure do not in themselves yield much direct evidence of the character of the function of a given area. A limited amount of such information is gained from the anatomic method by demonstration of the fiber supply to certain cortices, as, for example, the distribution of the relays from the retina to the calcarine area, but as a whole histologic studies merely give us data concerning the exact distribution in the mantle of different structural patterns while interpretations of the function of these areas depend for large part on correlation with data gained by the other methods.

All of the structural elements of the cortex show differences of arrangement in different parts of the mantle and we may therefore study the differences in cells, in fibers or in glia content and derive cytotectonic, myelotectonic or gliatectonic schemes for each zone. By far the most profitable of them is the cytotectonic, which takes into consideration the numbers, size and form of the nerve cells and their arrangement into layers and the number and relative thickness of these layers.

Brodmann studied the cortex from a large series of animals in the embryo, in the period of growth and in adult form, and while advancements and refinements have been added by others, his statements of the fundamental patterns are still widely accepted. Brodmann concluded that the basic type of lamination of the cortex of the neopallium of mammals is a six layered one and that all areas which have more or less than six are modifications of this fundamental arrangement. The archipallial cortices do not go through a six layered stage either in ontogeny or in phylogeny. The typical six layered type of the neopallium Brodmann called the homogenetic formation. Variants from this he called heterogenetic, and such heterogenetic patterns may be produced either by reduction, giving less than six layers in the finished pattern, or by splitting, giving more than six.

Several types of nerve cells are found in the cortex. The most characteristic and striking is the pyramidal or pyriform which is found in all cortices in varying numbers and size. Small, round or irregular nerve cells called granules or stellate cells are numerous in certain areas but are much more variable in occurrence and number. Fusiform and triangular cells are present in the lower levels of all areas, and in certain restricted fields large stellate or ganglion cells are to be seen.

The layers of the typical six layered cortex are as follows:

1. Lamina zonalis (molecular or plexiform layer). This contains very few nerve cells. It consists chiefly of myelinated and bare fibers of varying size and a supportive framework of glia. It probably serves as an important distributing zone for fibers originating in the underlying laminae.

2. Lamina granularis externa. This layer is a narrow zone of small nerve cells, and next to the internal granule layer is the most variable of the six.

3. Lamina pyramidalis. This layer is made up of pyramidal or pear-shaped nerve cells varying quite markedly in size. As a general pattern the smaller cells lie in the upper parts of the layer and as deeper levels are reached larger and larger cells are encountered, although there is also some commingling in size. The greatest size reached by individual cells of this lamina also varies from area to area and in some loci this difference forms one of the differentiating points. Campbell divided this layer into three—the layer of small pyramidal cells, the layer of medium sized pyramidal cells and the external layer of large pyramidal cells.

4. Lamina granularis interna (layer of stellate cells). This is the most variable of all the six layers. It is almost entirely lacking in the motor cortices and is strongly developed in all the arrival platforms and especially that of vision where it is divided into three layers. The fact that this layer of close packed small cells finds its greatest development in those areas in which the sensory paths first impinge on the mantle and certain other anatomic relations of the stellate cell has led to the view that this probably constitutes a distributing zone for incoming sensory stimuli.

5. Lamina ganglionaris (internal layer of large pyramidal cells). This contains in the main large nerve cells most of which are pyramidal in type; although in the calcarine area large cells of an irregular stellate form—the solitary cells of Meynert—are found at this level. This layer is looked on as the sending level of the cortex. Certainly this is true for the Betz cells of the precentral cortex and by analogy for the giant pyramidal cells of the postcentral and probably also for Meynert's cells of the occipital cortex. This layer is constant in all areas of the neopallium and in all animals.

6. Lamina multiformis. This layer varies strikingly in depth in different areas but is nevertheless constant in all areas and all animals. It contains a rather wide variety of cell forms as its name indicates—many of the cells are fusiform, others triangular and still others not unlike small granules. There is at times no sharp line of demarcation from the overlying ganglionic layer so that at their junction intermingling of pyramidal cells with the fusiform may be observed. Its lower border is often even less sharply marked, and scattered elements particularly of the fusiform type may often be found lying practically isolated in the white matter for considerable distances below their neighbors. Indeed, as the writer pointed out some years ago, the difficulty in deciding on the lower

border of this zone forms an important source of error in microscopic measurement of cortex depth. Many of these spindle cells have a considerable likeness to the wandering neuroblast, and the persistence of cells of similar form in higher laminae is beautifully shown in certain of Hammarberg's drawings of the brains of idiots. These facts and the frequent occurrence of fusiform cells scattered along the white fibers in the path which the migrating neuroblasts have followed to reach their cortical seats raises the question as to whether certain of them may not be looked on as later comers of the stream of neuroblasts which have lagged in their migration and which have failed to mature to the greater degree of differentiation shown by cells in the other layers.

Figure 2 is a diagrammatic sketch showing a homogenetic cortex arrangement and two types of heterogenetic.

The point at which each sensory pathway to the neopallium first debouches into the cortex is called the arrival platform and is characterized by a certain general plan of design though with marked modifications between areas of differing function. The zonal layer is moderately broad but not particularly characteristic. The external granular layer is constantly present and of fair depth. The layer of pyramids is variable in width and development but always well represented. The internal granular layer, or layer of stellate cells, is more strikingly developed in the arrival platforms than in any other area; indeed, it forms one of the landmarks of this type of cortex and yet it is also one of the most variable when areas of different function are contrasted with each other. In the calcarine cortex, for example, it forms a belt of great width subdivided into three distinct layers while in the postcentral field it is by no means so well represented, and yet even here it is better developed than in any of the cortices which are not of the arrival platform type. The ganglionic layer is constantly present, and large cells are to be found at this level in all the arrival platforms although there is striking variability both in depth of this layer and in the size and form of its constituents. By analogy with the Betz cell layer of the motor cortex this level is looked on as the seat of the sending cells for impulses relayed from this platform to other centers. The multiform layer is constant but variable. In the calcarine area it may be subdivided into two comparatively well marked layers.

Immediately surrounding the arrival platforms we find a second type of cortex in each functional zone of the neopallium which is variously described as the elaborative or "psychic" cortex but for which I have suggested the name recognitive for reasons which will be given later. These fields also, while often differing widely from each other, have certain general characteristics. They all show a well developed stellate layer which we have come to look on as a hall mark of sensory cortices but it is much less prominent than in the arrival platforms. The ganglionic layer is also less well developed. The general cell richness, the depth of the cortex and the size of its constituent cells is in the tem-

poral and postcentral fields much less than that of the arrival platforms. The occipital proves an exception to this only in the size of its cells.

A third general type occupies by far the greater part of the neopallium. The great posterior association zone, much of the temporal lobe and the anterior portions of the frontal lobe are covered by cortex

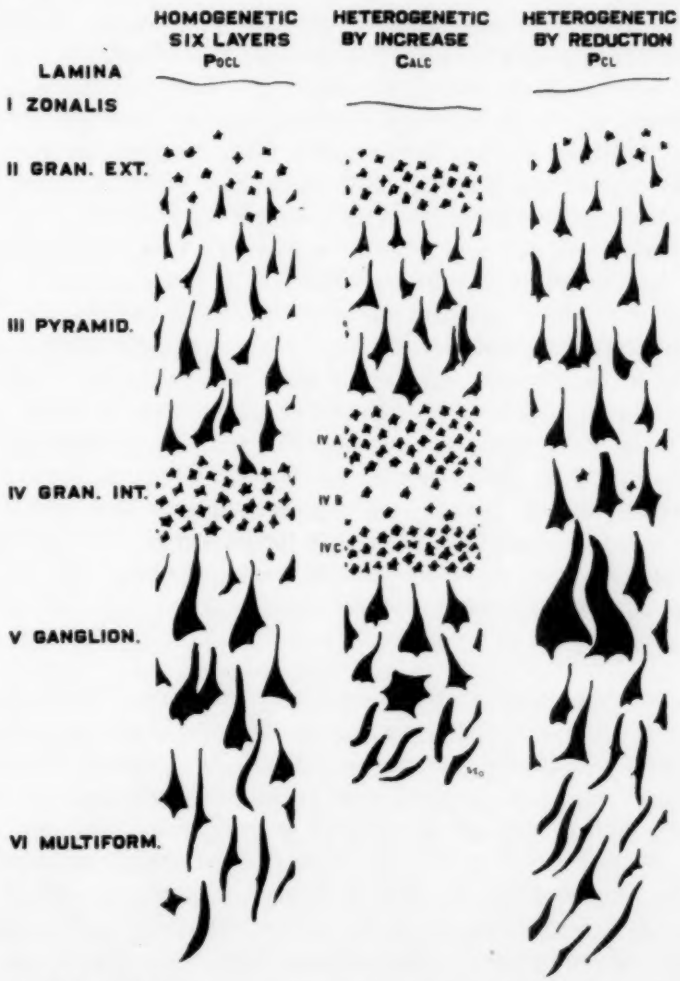

Fig. 2.—Schematic diagram of a typical six layered cortex, one in which the number of layers has been increased by fission and one in which reduction has occurred.

which also contains a fairly well developed stellate layer although consistently less broad than in either of the preceding types. Its depth is not so great and its cells are as a rule of distinctly smaller size, especially in the third and fifth layers.

A fourth type, the motor zone and the cortex immediately in front of it which Campbell called the intermediate precentral and to which Brodmann applied the name of agranular frontal is marked, as the latter name implies, by an almost total absence of the fourth layer—the lamina granularis interna—thus bringing these zones into sharp contrast with the sensory. The zonal layer in these fields is deep. The external granular layer is inconspicuous. The layer of pyramids is wide and contains a large complement of large cells. The internal granule or stellate layer is almost entirely lacking. A few cells of this type are to be found at this level in every brain but usually they are so few in number that they do not form a layer. Their number is, however, a variable in different persons. Brodmann has emphasized that in the embryo both the internal and external granule layers are well represented in the motor area and this lack in the mature cortex must therefore. I take it, be interpreted as the result either of a disappearance of cells of this type or of their later differentiation into the pyramidal form of the over and underlying laminae. Some of Hammarberg's illustrations give a hint of a much greater number of these cells in the motor cortex of the idiot's brain than in the normal and I have observed a similar picture. The fifth layer, the ganglionic, is deep and with a good complement of very large cells in all the granular cortices. The true giant cells of Betz are, of course, limited to the precentral zone but cells of great size are also to be found in this level in the intermediate precentral field. The multiform layer is constant and of good depth but not so strikingly developed as in the sensory cortices.

IV. The Myelogenetic.

The time of ripening of various functional areas of the cortex is a variable and the appearance of the myelin sheath on the nerve fiber may be used as a guide to maturation. Myelin is a product of the maturing neuron which is required for functional perfection but is not deposited until growth of the neuron is fairly complete. Before the end of the first month of extra-uterine life the medullary sheaths are deposited on the fibers of only a limited number of cortical areas. Flechsig and the Vogts and others have reported on this sequence. Flechsig outlined thirty-six areas differentiated by the time of myelinization which fell in general into three main groups—the primordial or early which have received their myelin by the end of the first month of postnatal life—which correspond with what we know as the projection centers, i. e., those centers which have the most immediate connection with the outer world, thus including the motor area and the sensory arrival platforms. The late or last group are those of the great frontal and parietal zone which are looked on as associative in function. The intermediate group are between the primordial and the latter, both in

time of maturing and in anatomic position and correspond with the elaborative cortices.

V. The Comparative Anatomic.

Some information of importance can be gained by the comparison of the structural relations of the brains of lower animals with that of man although this also is limited by the great functional difference represented in this step. Correlation exists, for example, between the relatively greater development of the archipallium in the osmatic animals and their olfactory acuity, and studies of the structures in these animals throws some light on the corresponding areas in man's brain. Again, man's brain stands out in sharp contrast to that of all other mammalia by reason of the much greater proportionate development of the frontal and parietal areas—the association zones.

The synthesis of an acceptable scheme of localization from the data gathered in various fields of investigation is far from complete, but such attempts have given us a working basis for further observation and experiment.

THE VISUAL CORTICES

The arrival platform for vision occupies a clearly demonstrable field around the calcarine fissure—this is the area striata which can be readily outlined even with the naked eye by the presence of the white line of Gennari. It forms a somewhat pear-shaped area beginning on the inferior lip of the common stem of the calcarine and parieto-occipital sulci and spreading out on either side of the calcarine sulcus posteriorly. It usually overlaps the occipital pole so that a small portion of it is visible on the lateral surface. This cortex is one of the most highly modified of the heterogenetic types. It is characterized by a wide stellate lamina which may be subdivided into three fairly distinct narrower layers and by a wide fusiform lamina which again shows evidence of splitting into two fairly definite layers. It may be taken as the best example of the development of a heterogenetic lamination by increase of layers through splitting.

In specimens stained by the Weigert method the line of Gennari stands out as a prominent bundle of myelinated fibers in the stellate layer. The area striata is the terminus of the projection path from the retina. At birth the only fibers of the occipital pole which have received their medullary sheaths are the optic radiations of Gratiolet which constitute the thalamocortical link of the optic path. In Weigert preparations of the baby's brain at this age they therefore stand out in high relief against the nonmyelinated background. This cortex is thus one of Flechsig's primordial zones.

The results of stimulation of this zone in animals as in all the sensory spheres are naturally of no value. Ablation experiments and

anatomicoclinical studies are, however, somewhat more instructive. Complete bilateral destruction of this cortex blocks the pathway for the arrival of any visual impressions into consciousness, the pupillary reflexes remain active showing that the retinal cells which control this response are still sensitive but there is a complete amaurosis to which the name of cortical blindness has been given. Because of the bilateral implantation of the macular bundle, however, this result follows only when the lesion is bilateral. The peripheral parts of the retina are not bilaterally implanted although there is crossing of fibers from the nasal half of each retina so that a unilateral destruction of the calcarine zone produces a homonymous hemianopia but with retention of macular vision. The occasional retention of central vision in cases with double homonymous hemianopia has not been adequately explained. One theory is that the macular implantation is widespread through the area striata and therefore only a total destruction of this type of cortex would produce total amaurosis. Another is that the macular bundle relays implant chiefly in the anterior portions of the area which are much less frequently damaged in vascular obliterations than are the more posterior portions. Von Monakow believes that part of the distribution of the macular bundle relays is to the second visual level, the visual recognitive cortex, and that thus even complete destruction of the area striata would not cause a loss of central vision.

When extensive bilateral lesions of the second level exist we might expect to observe the results of the function of the arrival platform brought into relief as it were by deprivation. Munk was the first to call attention to this condition which he observed in dogs after extensive ablations of the occipital poles and to which he gave the name of mind blindness. These dogs were able to direct their movements so as to avoid collisions with objects in their way but had lost all capacity to react to the meaning of visual stimuli. Thus, though they could find their way about, they did not recognize food by sight alone, did not cringe from threats with a whip, nor did they respond to the sight of their master. Odors and sounds brought adequate responses. The condition of mind blindness has since been recorded in man and is characterized essentially as was that of Munk's dogs. Loss of the arrival platform alone does not interfere with the mnemonic recall of visual images although it does produce a complete blindness. From these two points of view it would seem therefore that the chief part which the area striata plays in the visual function is that of determining the locus of external objects and in furnishing the element which differentiates external sensation from imaginal recall. Because of this determination of objective reality I have suggested the name of visuo-perceptive for this cortex in place of Campbell's term of visuo-sensory.

Immediately around the visuo-perceptive cortex and encircling that field everywhere except at its anterior portion is a fairly wide skirt of cortex strikingly different in structural pattern which conforms to that of the elaborative types; i. e., it is midway between the arrival platform type and the associative. The border between this and the arrival platform can be sharply outlined by the abrupt loss of the line of Gennari and the reduplicated stellate and multiform layers. It is not so clearly separable from other cortices at its outer borders. This area receives its myelin much later than the calcarine but before that of the parietal and frontal fields and hence belongs in Flechsig's intermediate group. Its period of myelinization apparently follows the maturation of the arrival platform. Lesions of this zone are practically never pure and extensive ablations without damage to adjacent zones are also practically impossible, so that we have no knowledge of the effect of destruction selectively restricted to this area. In cases with extensive bilateral losses in the occipital pole leaving only portions of the arrival platform intact we encounter, as outlined above, the condition of mind blindness. We again may see the functions thrown into relief when both of these lower levels are intact but a lesion at a higher level has resulted in losses of more complex visual functions. Thus with lesions of the visual associative area, sensation remains unchanged and there is correct appreciation of the objectivity of sensory stimuli and correct recognition of the concrete meaning of objects, but loss of the associative or symbolic meaning. To the second structural type which surrounds the arrival platform Campbell gave the name of visuo-psychic because we have come to associate this area with elaboration of the lower process. The term psychic, however, seems quite indefensible as applied to this restricted element of cerebral function and I have elsewhere suggested the name of visuo-recognitive for this cortex. Visuo-apperceptive approaches the psychologic terminology more closely but is a more cumbersome term and might readily be confused with the visuo-perception of first level.

At the outer boundaries of the visuo-recognitive field we find a more gradual transition zone than at its inner margins. The cortex of practically all the rest of the occipital pole (Brodmann's field number 19) is of this third occipital type but we have little or no exact information concerning lesions limited to this area. There is, however, a functional loss of a third order which bespeaks a third level of elaboration of visual stimuli, and it may be that these are to be correlated with this third structural field. This is the loss of the capacity for visual stimuli to arouse the corresponding abstract associations derived from other sensory fields. In psychologic terms, visual stimuli no longer arouse the corresponding concept although the concept itself is intact and may be aroused through other sensory paths. Because of the fact that words in

either the written or spoken form serve so generally as the medium of expression of symbolic or abstract meaning this loss is usually recorded in terms of word symbols, i. e., losses in the capacity for visual stimuli of written or printed words to arouse the concept or associative meaning, and such cases are usually recorded as examples of word blindness or visual aphasia. I know of no recorded cases but it seems highly probable that similar losses would occur in the capacity to understand the sign language of the Indians and the "finger talk" or lip reading of the deaf and dumb. Care should be taken here, of course, not to confuse the gestures of emphasis which carry a more objective and less abstract meaning.

A much wider experimental and observational program must be carried out before we can accurately associate this function with a deliminated cortical area. One important fact comes forward at this point, however, and that is that lesions productive of this syndrome need not be bilateral. The accepted location for lesions producing this condition is the left angular gyrus in right handed persons. This brings up the problem of unilateral dominance in the higher cerebral functions which will be discussed more in detail later. The angular gyrus, because of its strategic position at the junction of the occipital, parietal and temporal fields, has been considered as ideally located for associative processes but it also overlies a great mass of associative pathways linking these three zones, and the influence of deep lesions on these fiber tracts does not seem to have received sufficient attention. Without allowance for the influence of section of these pathways it seems incredible that this small zone could accommodate all the special functional elaborations which have been accredited to it.

THE AUDITORY CORTICES

The transverse temporal gyri of Heschel are the terminus of the third link of the auditory pathway. This area is one of Flechsig's primordial zones—receiving its myelin before the end of the first month of postnatal life, and its cortex shows the characteristics of the arrival platform type. This field occupies a protected position on the upper surface of the temporal pole in the sylvian fissure and is covered completely by the overhanging opercula so that traumatic lesions are not common. It is fed by branches of the sylvian artery but it shares this arterial supply with the insular region and first temporal gyrus and hence pure lesions by vascular obliteration of either this platform or the next are extremely rare.

Extensive bilateral lesions, which although not pure, yet have resulted in widespread destruction of both arrival platforms, are followed by complete loss of hearing of the cortical type which can be differentiated from peripheral deafness by the absence of hearing by bone

conduction. The arrival platforms, therefore, are looked on as an essential structure for the reception of auditory stimuli into consciousness. When extensive bilateral lesions of the first temporal gyrus exist but parts of the arrival platform are retained we see the function of this first level thrown into relief in the condition known as mind deafness which is quite analogous to mind blindness. In this the patient is aware of sound and of its direction of arrival but does not recognize the differential meaning of sounds so that the crow of the cock is not differentiated from the bark of a dog or the voice of a man. Thus by differential exclusion we are led to interpret the function of this area as a purely perceptive one.

The second area of the auditory group practically surrounds the arrival platform. It occupies the posterior two thirds of the exposed lateral surface of the first temporal gyrus and extends inward along the upper surface of the temporal pole into the sylvian fissure both behind and in front of the arrival platform. It is of the second or elaborative type and receives its myelin in the second or intermediate period. As outlined above pure lesions are extremely rare, and apparently, moreover, bilateral lesions are necessary to destroy its function. We have anticipated this function in order to bring into relief that of the arrival platform. In part by the results of widespread impure superior temporal lesions and in part by analogy with the visual function we have come tentatively to look on this cortex as the first elaborative zone for the stimuli of the audito-perceptive field, and we believe that here is added recognition of the concrete meaning of sounds. To see this function in contrast it is necessary to anticipate again that of the next higher order. With lesions in this higher level we meet the condition of word deafness in which the patient hears sounds, appreciates their objective nature and can differentiate their concrete meaning; i. e., recognizes a voice, but is unable to understand the abstract meaning of a spoken word.

The third order of function—the symbolic or associative—in audition as in vision can be clearly seen in the condition described above to bring the second level function into relief. This is the selective loss of the associative meaning of a spoken word without loss of hearing in the gross sense and without loss of recognition of the concrete meaning of sounds, which is called word deafness. The patient appreciates that he hears a voice but the spoken words fail to arouse the corresponding concept, i. e., it has lost its associative linkage, although the concepts, in pure cases, seem to be intact and can be aroused through other sensory channels. Differential losses within this functional level, such as are seen in the loss of a foreign language but with retention of the mother tongue, suggest a wide possibility of the association of different sounds with one concept.

No exact anatomic assignment can be made for this order of function but in a general way it may be said that lesions in the dominant hemisphere involving the posterior end of the first and second temporal gyri, thus approaching the angular gyrus, are followed by such losses. Again it is to be noted that at this level for the first time in the auditory elaborations a unilateral lesion is sufficient to destroy function.

THE EPICRITIC CORTICES

The cortex covering the surface of the postcentral gyrus and its two sulcal exposures—along the side of the fissure of Rolando and the postcentral sulcus—has been divided into two types by Campbell and into three by Brodmann. This area is the place of implantation of the thalamocortical relay of common spinal sensory paths. It is myelinated before the first month of postnatal life and hence forms a primordial zone. All of this area contains a wide stellate layer and hence is looked on as a sensory cortex. It is sharply demarcated from the motor cortex by the abrupt termination of the stellate layer which occurs at the bottom of the fissure of Rolando. The postcentral cortex contains large pyramidal cells in the ganglionic layer which are quite analogous in position and form to the Betz cells of the motor area. Their size is consistently less than that of the corresponding Betz cells, but those of the leg area, for example, are larger than the Betz cells of areas giving rise to fibers of shorter length such as those of the face area. The postcentral cortex bears a close relation by both position and arrangement to the motor cortex. Thus the epicritic area for each body portion is immediately behind the motor area for that same part. They are heterolateral in placement and arranged like the motor cell groups so that those highest on the vertex are linked with the lowest spinal centers. This cortex passes over the hemispheric margin at the central fissure and forms part of the paracentral lobule. Like the motor area it fails to reach the sylvian fissure by a short distance. The borders between the two subdivisions of Campbell and the three zones of Brodmann are not so well defined.

It is noteworthy that while we recognize the clearcut separation of tactile, kinesthetic and thermesthetic fibers and functions in the spinal cord there is only one area for the impingement of these sensations on the cortex. The recognition of the differential loss of the epicritic and protopathic fractions of these sensations has given a tentative explanation of this arrangement. The protopathic fraction—for example, the differentiation between touch and temperature—is distorted though not lost following lesions of the postcentral zone. The epicritic fraction—the capacity to localize accurately the source of such stimuli—is, however, destroyed, and we are therefore coming to regard this unitary field as organized for the recording and recognition of the epicritic or

localizing factor in common for all the spinal sensory paths while the recognition of the protopathic fraction and its consequent modifications of somatic reactions, etc., is resident in lower centers. Because of the very narrow bands of different cortical patterns here pure lesions of one structural type are not recorded. Extensive destruction results in complete loss of the epicritic sense in corresponding body areas, but here unlike either the visual or auditory fields a unilateral lesion is sufficient to produce a localized loss. This point will be discussed later. In this field we again see evidence, though by no means so clearcut as in vision and audition, of three functional levels. Thus we may have complete loss of the epicritic function which by analogy we would refer to arrival platform destruction. We also see differential loss of recognition of concrete objects. Such a patient is unable, with the eyes closed, to indicate by use his recognition of an object—a form of apraxia. In astereognosis the patient is unable to give the name of an object from its thermal, kinesthetic and tactile characters although he may be able to indicate its use. No exact anatomic localization can yet be given for the cortical residence of these three functional levels.

THE OLFACTORY CORTICES

The cortex serving olfaction is of the archipallial type and cannot, therefore, be closely compared with the neopallial patterns which have just been discussed. By analogy with the facts observed in lower animals and in the osmatic mammals we assign all of the archipallial cortices to the function of olfaction. The hallucinations of smell and taste which when present form so important a localizing sign in epilepsy are not usually associated with anosmia, even though the lesion be an extensive one, if it is unilateral. In extensive bilateral lesions of the cornu ammonis and hippocampal gyri anosmia is sometimes observed in epileptic patients. Loss of the olfactory sense without loss of imaginal recall of odors is also occasionally observed. I have recently had the opportunity to see briefly such a case. This was in a woman who fell down a flight of stairs, striking the back of her head on a cement floor. She was not unconscious but suffered a severe headache and considerable shock for several days. Immediately after the trauma she noticed that she could not smell spirits of camphor nor the odor of cooking onions. After a few days a parosmia appeared which was gradually subsiding. On examination she was entirely unable to recognize even strong familiar odors although she said each carried some impression to her. She said, however, that she at first could recall vividily what odors certain things should carry. This was becoming less vivid but she reported repeated disappointment in the flavors of foods she formerly liked.

THE POSTERIOR ASSOCIATION ZONES

A large part of the brain surface lying behind the fissure of Rolando is covered by cortex which shows minor local variations but which in general shows less special features in the way of variations in size of cells or in thickness of laminae than those already described. This type receives its myelin late and in general its largest cells are much smaller than those in the perceptive and recognitive fields. It also lacks special features such as the Betz cells and lack of granules in the motor zone, the cells of Meynert and divided granule layer of the calcarine, etc. It occupies roughly the parietal lobe and a part of the temporal lobe. Its functional significance is not clear and it forms part of the so-called silent areas because of the lack of accurate localizing symptoms following lesions here. Our clinical methods of studying such cases must here be challenged, however. Rarely do we have the opportunity to study the brain of a patient whose mental capacities and potential mental accomplishments have been critically studied before the onset of the disease, and for the most part our methods of study are designed to uncover only the grosser forms of defect in sensation and its elaborations. They are not yet refined enough to take into account the way in which the mnemonic material of past sensations is used in ideation nor the comparative facility of implantation of new ideational material. With the coming of such examination methods it seems probable that diagnostic advances in this field will be made, and the way seems prepared for certain progress in this direction. For example, the scope of the written vocabulary available to recall in a given person can often be fairly closely estimated from writings before the onset of disease and used as a measure to estimate reduction. Such vocabulary losses in gross form are, of course, readily observed in many organic dementias and in dementia praecox but I know of no studies of the finer grades of loss.

THE MOTOR CORTICES

The motor area proper, as already indicated, shows clearcut characteristics which make its delimitation quite easy. It shares with the intermediate precentral of Campbell or agranular frontal of Brodmann the striking lack of a stellate layer which differentiates these two fields from all the rest of the neopallium. The true motor cortex is further characterized by the presence of the giant pyramidal cells of Betz—the largest cells of the cortex. These cells are found not as a layer but as small clusters or nuclear groups in the lamina ganglionaris. Their distribution covers a wedge-shaped area of about half the width of the precentral gyrus below and extending over the stem of the first frontal sulcus onto the first frontal gyrus above and also overlapping the hemispheric margin where they cover all of the paracentral lobule in front of

the line of the rolandic fissure and where again they overlap onto the first frontal gyrus.

The axons of the Betz cells pass through the corona radiata and form the pyramidal tracts of the cord. There is, therefore, heterolateral representation here. Moreover, the lower spinal motor nuclei are connected with parts of the motor area which lie high on the convexity, and vice versa, so that there is a vertical inversion as well. The motor area matures shortly after birth and is hence a primordial myelogenetic zone. Lesions of the motor area result in man in permanent paralysis of voluntary motion without loss of reflex motion, upper motor neuron type of paralysis. A much greater degree of recovery results after lesions of this area in animals even as high in the scale as the monkey and this has led to some challenge of the current view of the function of the motor area. The increasingly greater prominence of the forebrain as the animal scale is ascended is paralleled by an increasing subjugation of the control exercised by lower centers and their progressive loss of autonomy. In man we find the peak of this process in which all voluntary motor control is apparently dependent on the integrity of this forebrain platform. Stimulation experiments have outlined very clearly the functional areas of the motor cortex so that our knowledge of the localization of special anatomic units, such as the hand and arm, within this cortex is fairly accurate. Unlike the nuclei in the spinal cord, however, the groups of Betz cells do not control the action of individual muscles or muscle groups but rather tend toward combining coordinated responses of related muscle groups which are resident in the lower centers. Sherrington has shown by his experiments with the spinal dog that many complex motor patterns are a function of the spinal cord and we must look on even such apparently purposive coordinations as the scratch reflex or the extensor thrust as the result of the structural interrelations of the anterior horn cells and of the afferent and internuncial pathways of the cord. These lower motor patterns probably serve as the material from which the motor cortices build the more purposive combinations which we call volitional motion.

The function of the agranular frontal field, that cortex which lies immediately anterior to the true motor zone, is not so clearly understood. It is strikingly unlike the cortex of any known sensory field and strikingly like the motor area in all its characters except that all of its cells and layers are constructed on a somewhat smaller scale and it does not contain any of the giant pyramids of Betz. Its time of myelinization places it among the secondary or intermediate zones. Stimulation experiments have given some responses and usually of a rather complex type as, for example, rotation of both eyes and head, but often they are negative over large parts of this area. Pure lesions here are extremely rare. A part of this area came early into prominence through Broca's

report of lesions of the foot of the third left frontal convolution in aphasics. Broca's area is covered by cortices which have been given separate rankings by Brodmann but is included in Campbell's intermediate precentral zone. Broca's views concerning this area have been seriously challenged, notably by Marie who claims that lesions here are not followed by aphasia unless there is extensive damage elsewhere. The view that this cortex serves as a place for the registration of "motor memory," i. e., the recall of motor patterns which have been learned, has fallen into disrepute. Its structure is quite unlike that of the areas which have been developed for the registration of engrams in the true sensory spheres; no clearcut clinical evidence of the existence of memories of motions divorced from kinesthetic and other sensory functions is forthcoming; and, finally, the loss of the capacity to carry out even such familiar motor patterns as speech and writing which follows destruction in certain sensory areas seems to indicate quite clearly that motor responses are slavish copies of engrams resident in sensory spheres. Tentatively the agranular frontal cortex may be looked on as a motor platform, of later development than the Betz cell area, probably having to do largely with the further compounding of the patterns which are resident in the true motor area, and hence playing a part in the synthesis of the more intricate movements such as writing, speech, playing musical instruments and athletic games.

THE FRONTAL CORTICES

The whole of the great frontal lobe, anterior to the two types of motor cortex just described, is covered with cortex in which the stellate layer is again to be observed. It may be subdivided into many smaller areas by refined methods of comparison of cells and fibers but except for these minor differences the whole field is occupied by cortex of about the same character. It forms the most recent phylogenetic development. Indeed, it is found well developed only in man and with the parietal zone forms the major difference between man's brain and that of the apes. Paralleling this late appearance in the phylum it is also an area of late ontogenetic development and is among the last parts of the human brain to mature. Again, like the parietal zone, it does not record the effect of small lesions in clearcut clinical pictures and is therefore included among the silent areas. Comparatively large lesions have frequently been reported without great modification of the behavior of the person but extensive destruction, especially if bilateral, is followed by a fairly definite syndrome which forms a fairly characteristic type of dementia. There is loss of ambition and energy, a striking lowering of the moral tone, a loss of judgment, an increasing difficulty in acquisition of new habits or even new motor acts and, as the process advances, a tendency to somnolence or torpor.

News and Comment

BRITISH—AMERICAN NEUROLOGIC MEETING

The invitation of the Section of Neurology of the Royal Society of Medicine for a combined neurologic meeting has been accepted by the American Neurological Association. It will be held in London, from July 25 to 28, 1927, inclusive.

The meeting of the British Medical Association will be held in Edinburgh, Scotland, from July 18 to 21, 1927, inclusive.

The program with the general outline of the meeting will be announced later.

ANNUAL MEETING OF THE ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

The next annual meeting will be held in the Hotel Commodore, New York, during the week between Christmas and New Year. A full program will be published in the next issue.

Abstracts from Current Literature

INTERMITTENT HYDROPS. HERMAN SCHLESINGER, Wien. klin. Wchnschr. **39**:68-72 (Jan.) 1926.

Intermittent hydrops is a swelling of the joints which persists for several days, recurs at regular intervals, is independent of definite inflammation, recovers and does not leave residuals such as heart lesions or anatomic changes of the joint. This affection, which has been recognized since 1845, is enigmatic, its symptoms are known but the cause is still a mystery. Up to the present time only about one hundred cases have been described in the literature. The picture is characteristic. The knee joint is generally affected, sometimes one and sometimes both. Occasionally there is swelling of other joints. In each of the author's nine cases the swelling was in the knee, and this is true of most of the cases so far described (of seventy-seven cases only one had no swelling of the knee). However, other joints were frequently also affected, as the hand and finger joints. There are no joints which may not become affected, not even the temporomaxillary and the vertebral joints. One of the author's cases occasionally had a swelling of the cervical joints synchronous with the hydrops.

The attacks usually run about the same course in each person, varying from a few days to several weeks. Paresthesia usually precedes the attack, but it may appear without warning. Five of the author's patients complained of severe neuralgic pains on the outer or frontal side of the thigh radiating from the hip joint. In one case pain always preceded the swelling by three days; in three cases it was simultaneous with the hydrops. In most cases the joint swells, and a feeling of drawing and weight exists which may gradually merge into a feeling of definite pain. About the second day (the height of the attack) the contour of the joint is entirely changed and it is very swollen. Because of the increase of fluid there may be a marked ballottement of the patella. There may be edema of the skin, sometimes it is unchanged, not noticeably hot, but it is generally pale. Heavy pressure on the joint does not seem to cause pain. In the beginning of the attack the patient often feels fatigued and indisposed. However, temperature changes are the exception. In a few of the author's patients the first attacks were febrile, but the later ones were not. From three to five days after the onset of symptoms the swelling disappears, the joint takes on its normal appearance and the function is fully restored. After the disease has endured for a longer or shorter time, there is usually a moderate atrophy of the muscles in the region of the joint.

Some patients suffer many years in exactly the same manner. One patient had attacks over a period of twenty-eight years, several others over a period of twenty years or more. Six of the author's patients had the peculiar "calendar symptom" habit, that is, they had dates marked in advance when they planned to be less mobile. In other patients the attacks occurred serially with intermissions of many months. The irregular occurrence of attacks has been observed and described, but in a minority of cases. The disease occurs more frequently in women than in men — seven of the author's nine cases were in women.

Infectious diseases of various kinds have been suggested as possible etiologic factors, but certainly they do not play a material rôle. Frequently

trauma causes an attack, but only in persons who are predisposed. Neuropathic patients are frequently affected. It has also been noted in some cases that an attack followed severe mental unrest or worry. Seven of the author's patients were distinctly nervous and two of them belonged to the manic-depressive group; one patient had hydrops only during a spell of depression.

An interesting observation is that in the families of patients there are other members afflicted with diseases belonging to the neurovascular group (asthma, bronchitis, hay-fever, etc.). The author is inclined to look on intermittent hydrops as a form of Quincke's edema and this opinion is accepted by Oppenheim, Cassirer and others.

The author has recently made the following unusual observations: (1) the intermittent joint swelling occurred in several members of two branches of the same family; (2) the hydrops was accompanied by Quincke's edema, the localization being typical of the acute Quincke's edema; (3) at least two members of this family, one with hydrops, suffered from hemophilia.

Blanc has described knee joint swelling in mother and daughter, and Dreyfuss in mother and son. The author's nine cases include five members of one family. Two had attacks of acute edema of the mouth and pharyngeal mucous membrane, and one suffered from bronchial asthma.

The author describes in detail the case of a woman who suffered from knee joint swelling associated with Quincke's edema and hemophilia for at least twenty years, and includes a diagram showing that a brother of the patient, aged 56, had rheumatism, a brother, aged 48, intermittent hydrops, a sister, aged 42, a questionable hydrops, Quincke's edema and asthma, a sister, aged 32, hydrops and hemophilia; a paternal cousin, aged 44, hydrops, and a daughter, hemophilia and hay-fever.

The relationship of intermittent hydrops to Quincke's edema is evident in the two sisters. Even though the edema of the mucous membranes did not always recur, it frequently was present with the joint hydrops. It appeared suddenly without apparent reason, reached great proportions in a short time and disappeared without leaving any trace.

Recurring edemas of the mucous membranes of the pharynx and larynx are described in the literature on Quincke's edema. The author has observed two patients who suffered from edema of the tongue. Another interesting case is that of a man, aged 50, who had profuse salivation with his attacks, and also at this time noticed edema of the parotid region. This substantiates the author's assumption that some cases of intermittent swelling of the parotid gland belong to the hydrops group. This patient also complained as follows: In the first years of his hydrops he had diarrhea, later, however, he had painful erections and profuse emissions though there was psychic impotence. The hydrops occurred regularly every thirteen days and continued for from three to four days and was always accompanied by the symptoms described. Various forms of therapy proved to be of no avail.

It is noteworthy that pregnancy and labor influence intermittent hydrops. In some women the attacks come on with the birth of a child and continue until the next pregnancy. A case is described of a patient who first had periodic swellings three months after the birth of a child. They continued two days, vanished and returned every seven days. During her second pregnancy, two years later, the attacks continued unchanged. However, six weeks after the birth of her second child, there occurred, synchronous with the hydrops, swellings of the ankles and the great toes on both sides, which ran the same course as the knee joint swellings. In three months there occurred

a third pregnancy when the swellings ceased and were absent for about a year. They reappeared seven weeks after the birth of the third child with slight variation as to location.

Two other cases of intermittent hydrops following the birth of a child are described in detail.

The causative factor of joint swellings is unknown. Cassirer suggests that a secretion and angioneurosis may be the cause of the swelling in Quincke's edema, and the author has accepted Cassirer's views for intermittent hydrops.

The appearance of hydrops following psychic emotion and the changes brought about by excitement during the disease process, argue in favor of Cassirer's theory. Also most cases observed are in nervous persons, some in definite psychopaths, and now and then in persons with organic nervous diseases. The familial cases are in families in which there are members suffering from nervous or vascular affections. Nervous symptoms often take the place of joint swellings. The patients are fatigued, unable to work or are very excitable.

In none of the author's nine cases did psychic influence prove beneficial, nor did psychanalysis prove effective in the cases in which it was tried.

There seems to be a relationship between intermittent hydrops and rheumatism. A brother of one of the author's patients had severe joint rheumatism, and one patient developed finger joint deformities after the hydrops had persisted for many years.

The author believes that it is difficult to explain intermittent hydrops without taking autointoxication into consideration. The attacks seem to have a certain analogy to periodic paralysis (Goldflam), to narcoleptic attacks and to paroxysmal tachycardia. They apparently belong to the large group of hydrops (acute Quincke's edema in all its forms). The author is of the opinion that in some persons toxic products are formed by intermediary metabolism which have an affinity for certain tissues and organs. An accumulation of these toxic products causes direct irritation of sections of the vegetative nervous system, or the substance enters the joint and brings about the hydrops.

The author further believes that the genital organs play an important part in the production of hydrops attacks and advances the following arguments: Joint swellings in most cases begin at about the age of puberty; cyclic joint swellings after the sexual decline in men are infrequent; the influence of pregnancy on the attacks.

The therapy for intermittent hydrops comprises a discouraging chapter. Occasionally, direct influence to the joint is brought about by massage, diathermy, compression, puncture, injection, washing out of the joint, roentgen ray, rest, etc., but these influences usually cause unpleasant reactions elsewhere, as other joints may swell. In some of the author's cases arsenic subcutaneously seemed to be of definite benefit, but was not effective in others. Many forms of medication and treatment have been tried — salicylate preparations, internal, external and intravenous, various forms of antipyrin, quinine, protein substances, radium, hydrotherapy, etc. Psychotherapy, electricity, vegetarian diet, etc., have also been tried, but the benefits derived have been transitory. The author suggests that sterilization with roentgen ray in both men and women might be curative, but his patients preferred the disease to the treatment.

In only two of the author's cases did the attacks cease: in one patient following the use of amidopyrine (pyramidon) (the preparation did not benefit

any of the other patients); in another patient after long residence in a high altitude. Undoubtedly the chances for spontaneous cessation of the attacks increase with age.

F. P. Moersch, Rochester, Minn.

A Pathologic Study of Dystonic-Dyskinetic Processes. W. J. Bernis, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **28**:338 (Nov.) 1925.

Seven cases were studied:

Case 1.—The clinical picture was that of repeated cerebral vascular insults with left-sided paresis followed by rigidity of the muscles, masklike facies, slurred speech, adiadokokinesis (according to Bernis, this was probably due to the general rigidity) and no evidences of pyramidal tract involvement other than absent abdominal reflexes. The case was apparently one of multiple cerebral softenings belonging to Foerster's group of arteriosclerotic muscular rigidity. The pathologic process was diffuse with a predominance of arteriosclerotic vascular changes; there were also some perivascular changes leading to a typical disintegration of cerebral tissue. The points of interest were: (1) the principal site of the lesion was in the anterior and middle portions of the striatum; (2) in spite of the extensive rigidity the substantia nigra was completely intact; (3) the absence of hyperkinetic phenomena in the presence of involvement of the large as well as of the small cells of the striatum; (4) the onset of the disease with a series of apoplexies, the principal lesions of which were apparently not to be found in the motor region (pyramidal system), so that it must be assumed that the focal lesions in various parts of the frontal lobe, and also perhaps those in the thalamus, were responsible for the early spastic pareses. The pathologic process was generalized, involving all parts of the central nervous system and especially the anterior portion of the thalamus and striatum. The clinical expression of this pathologic process was an akinetic-hypertonic syndrome. It was also noteworthy that in addition to the status cribratus and multiple encephalomalacia there was also to be seen a typical inflammatory process resembling that usually encountered in encephalitis lethargica, and, as Hohman has shown, in parkinsonism; in other words, there was a combination of cerebral arteriosclerosis and inflammation, and it was impossible to determine which of these was the determining factor in the production of the clinical picture in the case. The process was too diffuse to be of localizing value.

Case 2.—Clinically, the patient presented evidences of an arteriosclerotic dementia: vertiginous attacks and choreiform movements involving only the lower jaw and the left upper extremity. Here, too, the pathologic process consisted of severe arteriosclerotic changes in the brain, especially in the basal ganglia, and a more recent lesion, inflammatory in nature and limited to the caudate nucleus. The inflammatory process was in the right caudatum and the choreiform movements were on the left side. Here again one cannot be absolutely certain as to whether the inflammatory focus in the right caudatum was the anatomic basis for the choreiform movements; at any rate it is of no little significance that the latter were associated with a contralateral lesion in an area which is ordinarily held responsible for hyperkinesia. A more definite localization cannot be attempted. It is also stressed that in this case too, the large and small cells of the striatum were equally involved.

Case 3.—This was a severe arteriosclerotic process — status cribratus, especially of the putamen and caudate nucleus associated with a more acute inflammation of the meninges and brain substance. The inflammatory process

was not focal; this was true of the process in the parenchyma as well as in the glia, except for the lesion in the substantia nigra in which the most mesially situated cells on one side were found to be most severely affected. It was a combination of inflammatory and vascular disease of the brain. The vascular process affected every part of the central nervous system although the striatum, the hypothalamic region and the substantia nigra on the right side were more involved than the other parts of the brain. Ten years before this patient died she developed a hemiplegia which, according to the history, had improved rapidly; a cicatrix found in the internal capsule probably accounts for this vascular insult. In spite of the fact that the case resembled paralysis agitans closely and was marked by the presence of severe muscular rigidity, the globus pallidus showed little involvement; the striatum was far more disintegrated. Inasmuch as the substantia nigra was more affected on the right side than on the left and the tremor was more marked on the left side, one might perhaps be justified in attributing the latter to the lesion in this nucleus; in view, however, of Hohman's observations, Bernis believes that the lesion in the substantia nigra could only be held responsible for the muscular rigidity. Here again more precise localization is impossible.

Case 4.—In a case of severe muscular rigidity resembling that of paralysis agitans, the substantia nigra showed more involvement on one side than on the other; the basal ganglia also showed evidences of severe parenchymatous degeneration associated with irregular foci of arteriosclerosis. There was a strikingly severe reaction of the glia with definite knoblike formations and perivascular cell infiltration. Here again, there was a combination of a chronic vascular process with a perivascular disintegration, almost in the nature of an inflammatory process. The latter was more or less chronic in character, and as the entire disease lasted only three years, it cannot be excluded as the cause of the peculiar clinical manifestations. In spite of the fact that the tremor and the rigidity were more intense on the right side of the body, this difference in the symptomatology cannot be reconciled with the difference in the pathologic observations on the two sides, because although the lesions in the substantia nigra were more marked on the left side, the pathologic process in the other basal ganglia was of equal severity on both sides.

Case 5.—The brain showed typical changes of senility—marked arteriosclerosis with changes in the brain parenchyma; the oral portion of the caudatum and the adjacent portion of the putamen showed ventromesially a scar with evidences of a preceding softening. Clinically the patient showed evidences of a chorea localized to the face, the upper extremity and partly also the lower extremity. This localized chorea was probably due to the lesion in the caudatum. The brain in this case also showed evidence of inflammation, which, however, must be admitted, was not very marked. The distribution of the chorea, in view of the site of the lesion in the caudatum, would also seem to confirm the claims of Mingazzini and of the Vogts that the ventral portion of the striatum is the center for the head, the middle portion for the upper extremity and the caudal portion for the lower extremity. The case, however, presents nothing in support of the theory that hyperkinesia is an expression of destruction of the small cells.

Case 6.—The patient had a peculiar shaking of the upper extremities, brought out especially on intention; there was also slight rigidity of the right upper extremity. The substantia nigra showed relatively slight changes, although the cells on the right side were somewhat more affected; this may possibly account for the tremor of the right upper extremity, but as to this, Bernis is

not certain. Very striking, however, was the diffuse involvement of the gray matter of the cortex of the entire brain. As in all senile cases the lesions were more or less focal in nature with the greatest involvement of the frontal lobes. The brain also showed evidences of marked arteriosclerosis with the usual sequelae of such a process; in addition to this generalized process there was also to be found in the striatum, and in the striatum only, marked destruction of the ganglion cells, especially of the large ones; the cells of the globus pallidus were unusually well preserved. This case also presents great difficulties from a localizing point of view. The process was altogether too diffuse and too deep to be of any localizing value, at least in attempting to find an anatomic basis for the tremor.

Case 7.—This was a case of Little's disease with dementia, rigidity and contractures. Bernis believes that the case was one of fetal encephalitis with diffuse involvement of the cortex, possibly with syphilis as a predisposing cause. The unusually marked vascular changes at this age (the patient was 32 years of age) would perhaps substantiate such a hypothesis, although it must be admitted that the entire pathologic picture was not of syphilis of the brain. There was no marked status marmoratus; the basal ganglia were relatively intact; the pyramidal system was involved on both sides, though not completely; there was massive atrophy of the fibers with diminution in the size of the capsule and of the middle cerebral peduncle, the middle portion being apparently damaged most severely; the substantia nigra appeared hypertrophied; the pons was normal, although it may perhaps have been somewhat smaller in size. The case was striking clinically, because although the contractures could readily be overcome there were no clonic tendon reflexes. The presence of some pale areas in the thalamus may possibly have been responsible for the patient's general anesthesia and other sensory disturbances.

This entire investigation leads Bernis to conclude that various clinical pictures of the same character may have the same pathologic background as their basis, the symptomatology depending on the anatomic localization of the lesion whereas the course of the disease depends on the nature of the pathologic process. He also suggests the possibility of evolving some method for treating these apparently hopeless cases — hopeless on account of the arteriosclerosis —at least as far as the inflammatory component of the pathologic process is concerned.

Keschner, New York.

Treatment of Neurosyphilis by Malaria. Paul O'Leary, W. H. Goeckerman and I. Stephen Parker, Arch. Dermat. & Syph. **13**:301 (March) 1926.

"Fever therapy" has been practiced since 1887. Wagner von Jauregg first used Koch's old tuberculin. Then polyvalent vaccines of typhoid were used with good results and staphylococcus vaccines with poor results. With old tuberculin the remissions were noted to be more frequent, of longer duration and of greater degree. The value of antisyphilitic treatment after malaria is questionable. Kirschbaum and Koltenbach report a series of seventy cases of general paralysis treated with malaria as compared with seventy-eight not so treated. In those treated there were thirty-eight remissions as against only eight remissions in the second group. Only eight patients of the first group died as compared to thirty-three of the second group. However, the authors advise against making deductions too early. The serology does not become negative in all cases that show clinical improvement but does tend to become negative over a period of years.

The complications during treatment were loss of weight, weakness, anemia, epistaxis, herpes labialis, enlargement of the spleen, nausea and vomiting, jaundice, edema, uremia, urinary retention, enlargement of the liver, irritability, hallucinations and delusions, also the usual symptoms of malaria. Patients may have a relapse of malaria, but this is most often due to inadequate administration of quinine. There are three groups of these patients: (1) those in whom organisms can be found but without clinical symptoms; (2) those with very mild clinical symptoms, and (3) those with definite clinical symptoms but in whom the organisms cannot be found. All patients should have at least three negative smears before leaving the hospital.

The mechanism of the therapeutic action is not understood but several theories have been offered. It has been noted in general paralytic patients who died from some intercurrent fever with high temperature that it was impossible to demonstrate the spirochetes in the brain tissue at necropsy. It has also been noted that best results occur in those patients with the most marked malarial pyrexia. Goodall believed that, because of the close biologic relation between the plasmodium and the spirochete, the production of antibodies would be expected, but he was unable to prove this by experiments on rabbits.

York and Macfie believe that quinine kills many of the plasmodia, but not all; the dead organisms set free an antigen which stimulates the formation of immune bodies which kill the remaining plasmodia. Also that these immune bodies may account for the good results in general paralysis.

Certain precautions are advised with regard to the spread of the malarial infection in the community in which the treatment is to be carried out: complete isolation of the host during the course of treatment; the use of screens on the doors and windows where the patients are kept; isolation in places where mosquitoes are less likely to be found; the prophylactic use of quinine by those who come in contact with the patients; the covering of mosquito breeding places with oil; treatment of as few patients as possible when mosquitoes are prevalent.

Between July 1, 1924, and March 15, 1925, the authors inoculated thirty-five patients; twenty-four general paralytic patients, of whom twelve had simple dementia, nine were manic and three were emotionally depressed; four had "paresis sine paresi"; three were neurosyphilitic patients without symptoms who had received intensive antisyphilitic treatment; three were neurosyphilitic patients with optic atrophy; one was a patient with tabes, whose serologic examination gave negative results, but who had persistent lightning pains. The patients were inoculated by blood which was withdrawn from the infected host and injected intravenously or intramuscularly. The incubation period averaged seven days in those inoculated intravenously, and thirteen days in those inoculated intramuscularly. The first strain in this series has passed through thirty-three hosts without evidence of increase in virulence.

The results noted were: Of the general paralytic patients, six went into complete remission, nine were in incomplete remission within one month after malaria was stopped, three were uninfluenced and two died (one from malaria and one from general paralytic convulsions); four had not been heard from. Of the six with complete remissions, two had had previous remissions after treatment with tryparsamide; the remaining patients had had none. The nine who were improved had no history of spontaneous or induced remission, while the three who showed no change were at the time in a state of partial remission. The asymptomatic neurosyphilitic patients with positive serology showed no

immediate change. One of the cases of "paresis sine paresi" showed definite improvement. The three patients with optic atrophy were made worse immediately after treatment, but they returned to the previous condition within a few weeks. No change was noted in the case with lightning pains. Very little change was noted in the serology of any of the cases. In the general paralytic patients the results were better in those in whom the disease was of short duration.

The complications noted were mainly due to the malaria. Thirteen patients were delirious at some time during the treatment; a few had hallucinations and delusions but these symptoms disappeared with the cessation of the fever. Progressive weakness was noted in almost all cases, and was so severe in four as to necessitate the cessation of treatment early, but recovery in these cases was rapid. Loss of weight averaged 8 pounds (4 Kg.), although two patients showed a definite gain during treatment and practically all gained after treatment. Nausea and vomiting, diarrhea, rectal and vesical incontinence, urinary retention and varying degrees of edema were noted. Anemia was a constant complication, but this also disappeared rapidly after the malaria was controlled. A few cases showed a lighting up of the gastric crises, and a few showed an exacerbation and increase in the severity of the lightning pains.

During the course of the malaria a careful check was kept on the blood urea and the percentage of red cells that contained plasmodia. The patient who died had a very high blood urea. The percentage number of plasmodia apparently had no bearing on the severity of the toxic symptoms.

The treatment of the malaria was as follows: 5 grains (0.3 Gm.) of quinine twice daily; then one week without quinine, a second week of quinine treatment followed by another rest of one week; quinine was also given during three days of the fifth and seventh weeks. The total amount of quinine given consisted of forty 5 grain capsules in seven weeks.

WAGGONER, Philadelphia

INFLAMMATION OF THE CENTRAL NERVOUS SYSTEM. DONALD J. MACPHERSON. Arb. a. d. Neurol. Inst. and Wien. Univ. **28**:283 (Nov.) 1925.

The object of this investigation was to determine whether certain degenerative processes in the central nervous system, in which one finds no evidences of exudation, can be designated as "inflammatory" or whether it would not be better to designate them as "pathies" in the sense that Aschoff employs this term. Histologic examination alone could not settle this question, and the author was compelled to seek its solution in a series of experiments. The first part of the problem was to determine under what circumstances exudation occurs and what conditions in the organism itself influence its occurrence. To accomplish this it was necessary to bring about a diminution in the acid content of the blood in order to see whether or not this would produce an alteration in the exudative process. For this purpose MacPherson administered, according to the method of Mansfeld and Müller, aqua amygdalae amarae to guinea-pigs in such doses that, although poisonous, the animals could still survive. This procedure caused no perceptible difference in the hemoglobin content of the blood. (It must be borne in mind that poisons which reduce the hemoglobin content of the blood also produce, directly or indirectly, changes which influence cerebral processes.) To another group of animals poisonous doses of phenyl-hydrazin were given; these caused definite diminution of the hemoglobin content (50 per cent). In a third series of animals the thyroid gland was removed by thermocautery. In the surviving animals of each group

the brain was traumatized under aseptic precautions and examined in serial sections. The tissues showed different reactions in each. Whereas the animals that had not been previously poisoned showed a reaction in the connective tissue as well as in the glia (most marked in the latter), the poisoned animals showed a reaction which was far more intense in the connective tissue. In the poisoned animals there was found, within a few days after the trauma to the brain, an intense proliferation of the connective tissue cells extending from the pia and blood vessels and filling up the scars. Neither of these animals, however, showed any hemorrhage or softening of the surrounding glia. As far as the glia was concerned, considerable differences could be observed in the animals poisoned with aqua amygdalae amarae as compared to those poisoned with phenyl-hydrazin. In the former large protoplasmic glia cells appeared which apparently were devoid of any fibrillogenetic function—evidence of marked degeneration; it is not at all unlikely that here one had to deal with ameboid (dysplastic) cells or elements similar to them. It was impossible to determine the origin of the large number of glia cells observed in these cases. It must be assumed that hematogenous elements play an important rôle here, because in the animals poisoned with phenyl-hydrazin the lymph sheaths of the adjacent vessels were filled with an unusual number of round cells. The very large glia cells were also to be seen in the animals poisoned with phenyl-hydrazin, but they were much fewer in number than in the animals poisoned with aqua amygdalae amarae. In conformity with the changes in the glia cells the ganglion cells also showed evidences of severe injury— degeneration in the form of vacuolization; the granular cells themselves were also degenerated. There was marked difference between the non-poisoned and poisoned animals in that the intensity of the glia reaction in the latter was much more marked than in the former, and also that in the former the reacting glia cells appeared dysplastic. The thyroidectomized animals behaved more like those poisoned with phenyl-hydrazin; unfortunately the former did not survive the experiment very long; this may perhaps account for the fact that in these animals the connective tissue changes were so marked.

It is, therefore, quite evident that these experiments do not shed much light on the problem that the author attempted to solve. Tissue reactions were found under normal as well as under pathologic conditions. At the same time it was also found that the expected injury to the tissues did not materialize, so that one really has no right to speak of an exudation from the vessels. The most manifest feature was an injury to the tissues due to poisoning. As, however, there is no better index of reaction on the part of the central nervous system than the condition of the neuroglia, it must be presupposed that for this component of inflammation, i. e., reactive glial proliferation, the existence of an acid-rich blood is a sine qua non, provided, of course, that the poison is not administered in doses sufficiently large to produce direct injury to the glia. MacPherson hopes to shed more light on this problem after he has devised a method of experimentation that will enable him to exclude the direct effects of the poison on the tissues.

KESCHNER, New York.

CONTRIBUTION TO THE HISTOCHEMISTRY AND PATHOGENESIS OF CHARCOT'S DISEASE. G. MARINESCO, Rev. neurol. **2**:543 (Nov.) 1925.

The author endeavors to establish that the morphologic lesions of Charcot's disease are the expression of a breaking down of the physicochemical structure of the neuron, and that the lesions which occur in the altered colloidal medium

of the nerve cells are the product of abnormal activity of intracellular ferments. These cells possess a different colloidal structure, and varying amounts of oxidizing ferments and catalyzers. This variability of structure explains the localization of Charcot's disease primarily in the cells of the anterior horn and in the pyramidal tracts at the level of the synapses. The forces that give rise to active phenomena at a low temperature, without great differences in the alkalinity and acidity of the cells, are the oxidizing ferments and catalyzers. These latter substances, in the form of zinc, manganese and iron, are present in all cells. The ferments are the cause of all biochemical processes: hydration, dehydration, synthesis, etc. As to the existence of catalyzers other authors seem to agree; but in regard to the existence of ferments opinions are divergent. According to Marinesco, the oxidizing ferments act on the cytoplasm, while the catalyzers act dominantly on the nucleus.

From the pathologic point of view, the lesions of amyotrophic lateral sclerosis, especially the chromatolysis, must have a biologic significance, since according to Marinesco this is nothing else than the chemical disintegration of colloidal particles. According to Soula, chromatolysis is the result of proteolysis, so that the functioning of nerve centers is accompanied by destruction of albumin, the contrary of what happens in muscular tissue, the functioning of which is connected with hydrocarbons and lipoids. In the nervous system, the further advanced the proteolysis the more ions are liberated, among them the iron ion which leaves the colloidal granulations with the consequence that none of this metal is detected in the chromatolytic zone. By appropriate methods it can be shown also that the oxidizing function has disappeared from the chromatolytic region. Marinesco believes that the disturbances of the oxidizing function result from modification in the acid-base equilibrium of the cell owing to toxic agents. The nerve cells control the acid-base equilibrium of the nerve and muscle fibers, so that the cellular lesions correspond to hyperactivity of the hydrolytic ferments, which accelerates the decomposition of proteins and lipoids.

As regards the seat of origin of disease, Marinesco believes that lesions affect simultaneously the peripheral and central neurons, and that they begin at the level of the synapse which is a locus minoris resistentiae, and which contains a great amount of the oxidizing agents. The lesions of the cortical cells are of the secondary type. The pathologic changes, which at first are synaptic later become transsynaptic. The lesion of the peripheral nerve zone affects primarily the nerve cell.

From another point of view, Marinesco compares the lesions in Charcot's disease with those seen in vitro due to autolysis. Among other similarities he claims that the pigment of the degenerated nerve cells is homologous with the granules which are seen in the ganglion cells in a state of autolysis. These pathologic, morphologic and chemical changes of the nerve elements are due to the action of autolytic ferments, which exist in nerve fibers and nerve cells, and which display their action when the nerve fibers are removed from the control of the nerve centers. The hydrogen ion concentration must play an important part in the degenerative process as it does in the autolytic process.

Concluding from the pathogenetic point of view, Marinesco sees in amyotrophic lateral sclerosis, as in other familial and constitutional diseases, a disturbance of the reaction of the cellular medium which favors the action of certain ferments that reduce and even inhibit the action of the oxidizing and synthetic ferments. In this way the hydrolytic ferments increase their activity, the proteins are decomposed and the reaction of the cellular medium is modified.

The activity of the ferments is progressively reduced, intracellular combustion diminishes and the acid products which accumulate in the cells hinder the normal cellular metabolism.

FERRARO, Washington, D. C.

POSTURAL AND RIGHTING REFLEXES (STELLREFLEXE) IN DELIRIUM TREMENS. HANS HOFF and PAUL SCHILDER, Jahrb. f. Psychiat. u. Neurol. **44**:189, 1925.

In a previous communication these authors had pointed out that with the disappearance of the delirium postural and righting reflexes are diminished or entirely absent. Since then they had had an opportunity to study eleven more cases of delirium. They found that during the delirium these reflexes were increased, especially the turning reflex; in one case they even observed spontaneous lateral deviation of the upper extremity. In this group they also found that, following the delirium, the postural and righting reflexes were almost regularly diminished. In five cases they obtained no reactions at all; in two there was no difference in height between the "chin arm" and the "occipital arm," but there occurred a deviation-turning reaction. One case was interesting because during the delirium the reflexes extended from the lower to the upper extremity; this type of reaction they never observed in normal cases nor in any of their other cases of delirium. The authors attribute this type of reaction to a change in the mechanism of muscle irritability on account of the patient's badly mutilated hand (almost all fingers were gone); except for this anomaly all other reactions of the upper extremities were demonstrable, and they all disappeared with the subsidence of the delirium. It was also of interest to note that in two cases the reactions persisted, and became more intense from five to six days after the disappearance of the delirium. Normal reactions were also found among the delirious patients as well as among the cases of chronic alcoholism.

In explanation of these phenomena it must be borne in mind that lesions in the cerebellum inhibit postural and righting reflexes. There is also every reason to believe that during delirium the cerebellum is affected, so that the centers for these reflexes, extending, as they do, from the medulla to the red nucleus, may also be irritated during delirium. This hypothesis finds support in the well known fact that during delirium the nuclei of the ocular muscles are affected. With these facts in mind the diminution and absence of the postural and righting reflexes after the disappearance of the delirium could be explained as a paralysis (exhaustion) following a transitory irritation of these centers.

These observations are significant because they tend to establish an important analogy between epidemic encephalitis and delirium tremens. The authors had occasion to observe two fresh cases of epidemic encephalitis with delirium, both of which showed in the acute stage a marked increase of the postural reflexes, and especially of the turning and deviation reflexes. As has previously been pointed out the absence of these reactions is unusually common in parkinsonism. As a matter of fact these reflexes were absent in many cases that showed little or only slight muscle rigidity. At the same time it must also be admitted that there may occur cases of parkinsonism with perfectly normal reactions, and, here and there, one encounters a case in which there may even be an increase in the intensity of the reactions. These variations are attributed to the nonuniformity of the localization of the pathologic process in the different cases of parkinsonism.

KESCHNER, New York.

THE COMPARATIVE METHOD IN PSYCHIATRY. WILLIAM A. WHITE, J. Nerv. & Ment. Dis. **61**:1 (Jan.) 1925.

Psychiatry, like medicine in general, is an art rather than a science but utilizes the material of many sciences. For it the two forms of pathology—neuropathology and psychopathology—constitute the sciences most characteristic of psychiatry. Just as all sciences have passed through a stage of collection, description and classification of their material, so psychiatry is a body of scientific facts still largely in the descriptive stage of development. Its interpretive stage must come later. Just as perception of similarities is a process in the evolution of the human mind, so in science it becomes a preliminary stage of development and may be termed the comparative method. It is an approximation to the laboratory ideal. Anatomy, morphology, embryology, have all developed thus. The comparative method in the study of functions of the nervous system is exemplified in the work of Rosett in epilepsy and of Hunt on the corpus striatum. Only recently has psychology emerged from traditional medievalism to become a biologic science, and in genetic and animal psychology it has utilized the comparative method. In psychiatry, however, the progress has been slower and it remained for the psychanalysts to insist on the need for the historical study of the symptom. Hitherto, the content of thought and the sensory and perceptional aspect of the mind, rather than the motor and expressive aspects of thought that have been carefully studied by the comparative method, have been confined to the study of human speech, in which study we can draw distinct differences between primitive and civilized language, as well as an analogous difference between the speech of the child and that of the adult. In both the child and the savage concrete expression has not yet been replaced by the abstract. As Jespersen has said, "The evolution of language shows a progressive tendency from inseparable irregular conglomeration to freely and regularly combinable short elements." The savage as well as the child shows in his thought relative simplicity and lack of differentiation not only of his environment but of himself from his environment. The world of both is concrete and perceptional.

Taking schizophrenia as the major problem of psychiatry today, we can discern in the schizophrenic person an approach to the type of childish or primitive thought. A loss of objectivity, imperfect distinction between hallucinations and reality, and a preponderance of feeling function over reasoning function characterize both the schizophrenic person and the primitive savage. The world of reality has lost its identity as something separate from him. The primitive idea of forces passing through space from one object to another is similar to the schizophrenic weaving of his personality, his own autistic world, with the world about him. This patient's sense of individuality is lost and he feels a sense of loss at every contact with his environment. Autistic play, neoplastic language, perseveration and stereotypy, and a living more in the unconscious or subconscious self is characteristic of both the child and the schizophrenic person—and in the latter is looked on by the author as founded probably on incomplete constitutional development with defects of the ductless glands.

By this use of the comparative method in the great psychiatric problem of schizophrenia, the author suggests how further understanding of the human being may be possible through a recognition of the organism as a whole, as an outcome of millions of years of experience integrated by the nervous system—with, therefore, of necessity many archaic components.

HART, Philadelphia.

QUANTITATIVE MEASUREMENTS OF THE EXCITABILITY OF THE CENTRAL NERVOUS SYSTEM AFTER THYROIDECTOMY AND THYMECTOMY. F. C. NEWTON, Am. J. Physiol. **71**:12 (Dec.) 1924.

Newton was led to this investigation by certain observations that suggest a decrease in nervous activity in hypothyroid organisms and an opposite condition in hyperthyroid states. Of more direct relationship and serving as the actual stimulus for the present study was the observation by Rutchi that respiratory frequency was increased much less in response to temperature elevation in thyroidectomized animals than in normal animals. Rutchi also noted that when the thymus was removed as well this decreased excitability of the respiratory center with reference to heat became more marked and apparently permanent. Newton's own objective was the actual quantitative determination of these relations discovered by Rutchi.

Newton's apparatus consisted of an asbestos-covered box into which the experimental animals (rabbits) were placed. This asbestos chamber was electrically heated and so constructed that all external light and movement and practically all sound could be excluded. The respiratory movements were kymographically recorded by means of two rubber tambours adjusted to the subject's thorax, the animal being tied on a board within the warming chamber. Through preliminary work it was found that four minute temperature exposures gave typical and optimal results and that the most satisfactory temperature range was from 20 to 35 C. The actual experimental schedule was, therefore, formulated on this basis.

After the normal relation of respiration rate to temperature change had been determined, the animals were thyroidectomized and on recovering from the effects of the operation were retested to temperature. After thyroidectomy there was noted a definite and consistent lowering of respiratory rate in response to temperature elevation as compared to the preoperative reaction. From these same animals the thymus was now removed and after operative recovery they were studied again with reference to the respiration temperature relation. In these thymo-thyroidectomized subjects, the respiratory response to increased temperatures was diminished much more as compared with the normal response than when only the thyroids were removed. The increase in respiration rate occurred much more slowly and attained only one-half the rate for normal animals and only two-thirds that determined following thyroidectomy, as shown in the accompanying table. In response to morphine

	Average Rate of Respiration per Ten Seconds to Temperature Elevations (Adapted)														
Temperature, C.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
Normal animals	7	8	9	15	21	23	25	28	34	37	43	47	47	47	48	47
Without thyroid	6	8	10	11	14	15	18	19	21	23	27	30	31	31	32	34
Without thyroid and thymus	6	6	6	6	6	7	7	8	9	10	12	14	15	18	20	22

sulphate intravenously administered, it was found that the respiration acceleration with temperature rise was definitely diminished in the thyroidectomized animals and practically abolished after both thyroidectomy and thymectomy.

These observations lead the author to the inference, substantiating previous observations, that removal of the thyroid and thymus decreases the excitability of the central nervous system to environmental stimuli, and also that there appears to exist an "augmentary interaction" between these two glands in relation to the functional activity of the central nervous system.

RAPHAEL, Ann Arbor, Mich.

THE PATHOLOGY OF CEREBRAL CHOLESTEATOMAS. TAMAO KAKESHITA, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **28**:327 (Nov.) 1925.

Two cases of cerebral cholesteatoma were studied histologically. In the first case the pathologic cells resembled closely those found in arachnoidal endothelium; they were identical with those usually observed in endothelioma of the dura. They were increased in number, and, in scarlet red preparations, could be seen to contain a staining substance which resembled lipoid or was actually a form of lipoid material. Here and there the granules were brownish rather than red or orange, but inasmuch as the preparation had been kept unusually long in formaldehyde, this change in color may possibly have been due to the formaldehyde. None of the staining methods, however, showed any intracellular kerato-hyalin bodies. It was also striking that wherever the tumor assumed the typical characteristics of a cholesteatoma, the whorls also showed typical corneation. This complicates the problem because it is difficult to understand how lipoid substance can give rise to corneal tissue. The transition beween these two types of tissues seemed a gradual one; there were evidences of intermediate stages of cell formation varying from well preserved cells to severely degenerated areas containing lipoid material and finally to a cholesterin-containing network. This observation seemed to point against the idea that one might perhaps be dealing with two coincidental, different cell formations but that one of these could not be actually demonstrated.

The second case was that of a distinct endothelioma of the dura, almost sarcomatous in character. In this tumor it was quite evident that the cells had first taken up the lipoid material after which they were destroyed, whereas the connective tissue network which had contained these cells remained intact. This specimen also showed that cholesterin is formed from lipoid. It is questionable, however, whether in addition to the tumor cells the tumor does not also contain some lymphoid elements, in which, as in macrophages, there are to be found deposits of lipoid substance. Another significant observation in this case was the almost complete isolation of the cholesteatoma from the tumor mass; at least this was true in the sections examined — there being no evidences of a transitional process between the two. The only positive evidence of the relation of the cellular tumor to the cholesteatoma was the necrosis within the tumor tissue which began to show within its center signs of a beginning cholesteatomatous process.

The histologic interpretations in these two cases would seem to give an entirely new aspect to the usual conception of cholesteatoma, which is that of a special kind of tumor — an epidermoid in the sense of Boström and Erdheim. Apparently Boström's original views still hold true for some of the cases, but it would also seem that some forms of cholesteatoma exist that originate from endothelioma. The latter group might be placed in the same category with the psammomas, because after all, the psammomatous character of a tumor is merely determined by the admixture of psammomatous bodies with the original tumor. So that, in addition to the usual genuine cholesteatoma, which is an epidermoid tumor, there also exists a so-called "symptomatic cholesteatoma" which may occur in an endothelioma, or a cholesteatoma-endothelioma, similar to psammoma-endothelioma. What relation these cases have to plexus cholesteatomas, Kakeshita does not know.

KESCHNER, New York.

TENTATIVE THERAPEUTICS IN EPIDEMIC ENCEPHALITIS. L. VAN BOGAERT, J. de neurol. et de psychiat. **11**:708 (Nov.) 1925.

Specific treatment of epidemic encephalitis is of course still lacking, but to refuse to try tentative methods of treatment is to deny aid to the victims of a most terrible disease. Carnot and Blamoutier, in 1923, first used intravenous injections of salicylates in large doses in cases of encephalitis. They reported observations on two cases, one a case of acute chorea which was relieved by treatment. They used a 10 per cent solution, but because of the danger of obliteration of the veins they advocate a 4 per cent solution. This therapy was tried by Rothesy and Gournay, Benard, Marchal and Bureau and others who reported many successes. Since 1923, the author has used salicylates in combination with urotropine in the treatment of epidemic encephalitis. All the observations were made on patients who were seen early, many in the febrile phase of the disease, and many from the very onset, and Bogaert believes the early treatment has played an important rôle in the results obtained.

Seven cases of acute epidemic encephalitis are recorded with the following clinical pictures: acute severe chorea with respiratory troubles, ocular form, akinetic-myoclonic form, akinetic-lethargic type (two cases), polyneuritic type and akinetic type. Of the seven patients, five were distinctly relieved and one has had no trouble for two years. The influence of the medication is complete both on the clinical pictures and on the temperatures. Suspension of treatment causes relapse. Bogaert concludes: that the akinetic forms are more susceptible to treatment and the hyperkinetic more resistant; that there are no cases of encephalitis in which the salicylic-methenamine treatment, when instituted early, does not give some results. Three cases were uninfluenced by treatment: two lethargic cases with delirium and severe chorea, and one of generalized myoclonus. Of the drugs given the sodium salicylate seems the most efficacious because in two cases when given alone it gave good results. Bogaert advocates the use of the methenamine, however, because of its anti-infection properties and its permeability. The dosage employed varied from 2 to 8 Gm. of salicylate and 1 to 4 Gm. of urotropine daily; it is important to guide the dosage by the severity of the symptoms and the temperature. In the severe cases the minimal dose has been from 4 to 5 Gm. daily, though Carnot and Blamoutier gave 50 Gm. in one of their cases. The urine must be watched carefully for a microscopic hematuria which subsides in from twenty-four to forty-eight hours after the cessation of treatment. Symptoms of intolerance to salicylates are not frequent. In from three to four hours after the injection headache is common with some ringing in the ears, palpitation and nausea. Doses of from 20 to 25 Gm. daily by mouth are insufficient; the intravenous method is the method of choice in these cases.

ALPERS, Philadelphia.

ON THALAMIC AND TECTAL NUCLEI AND FIBER PATHS IN THE BRAIN OF THE AMERICAN ALLIGATOR. G. CARL HUBER and ELIZABETH CAROLINE CROSBY, J. Comp. Neurol. **40**:97 (Feb.) 1926.

This paper of 131 pages is a continuation of Dr. Crosby's memoir on the forebrain of the alligator (*J. Comp. Neurol.*, 1917). The authors here give the first detailed account of the internal structure of the reptilian thalamus and midbrain. The importance of this subject lies in the fact that in human neurology it is difficult to separate the functions which are proper to the

thalamus itself from those which this region shares with the corpus striatum and cerebral cortex. In reptiles the cortex is just emerging and the striatum complex is very elaborately differentiated.

The aim of this paper is to show in detail for reptiles the interrelation of cerebral hemisphere and thalamus and of thalamus and midbrain. It is based on serial sections of many brains prepared by the methods of Cajal, Weigert, Nissl and Golgi. The results cannot be summarized here, but a few points of general interest may be mentioned.

The midbrain is very elaborately developed and from its roof strong fiber tracts pass to the dorsal thalamus (nucleus sensitivus). In the *Amphibia* these connections are present, but relatively small, for most of the tectothalamic fibers reach the ventral thalamus (subthalamus) and hypothalamus. The amphibian dorsal thalamus is a very undifferentiated nucleus diffusus. In the alligator many special nuclei are separately organized, but this arrangement has not reached the mammalian pattern.

The mammalian nuclei of the medial group are well represented in the alligator. These nuclei are connected with the tectum and corpus striatum. The anterior nucleus is not clearly separated, but its primordium is recognized. The lateral (somesthetic) group of nuclei also are not developed in mammalian pattern, though their precursors are obscurely evident. The lateral geniculate body is well developed. It is connected by strong fiber tracts with the optic tectum, but no ascending optic radiations to the cerebral hemisphere could be demonstrated. Thalamostriatal, striothalamic and striotegmental tracts are well developed. No sensory radiations passing directly from thalamus to cerebral cortex have been definitely observed; most of these fibers pass to the striatum complex, from which they may be relayed to the cortex.

HERRICK, Chicago.

TREMOR OF THE EYEBALLS DURING CONVERGENCE. VLADIMIR VUJIĆ, Jahrb. f. Psychiat. u. Neurol. **44**:163, 1925.

A far advanced general paralytic patient, aged 37, was subjected to malarial treatment which was followed by considerable improvement. Six months later when he presented himself for the routine "follow-up" examination he showed: dementia; disturbances of memory and attention; dysarthria; ataxic handwriting; dilated pupils, left more than right, both being irregular in contour and stiff to light with slight reaction in accommodation in the right, and none in the left. All extra-ocular movements were well performed, and there was no diplopia, but on approaching the fixation object there was noted at a distance of about 15 cm. from the latter a latent external squint (it is not stated in which eye). On looking to the extreme right and left the eyeballs showed an occasional nystagmoid twitching. On testing for convergence, a slow approximation of the fixation object was promptly followed by perfect convergence, but on approaching the object very rapidly there suddenly appeared in both eyes (simultaneously) an exquisitely fine and rapid horizontal oscillation of both eyeballs. As far as one could determine with the naked eye, the to-and-fro components of this oscillation were equal in rapidity. The great speed of the oscillations made it impossible to ascertain whether these were in the same or in the opposite direction of the visual axes. The number of oscillations was approximately 1,000 per minute; they persisted as long as the patient's eyes remained fixed but it was impossible for him to keep his eyes fixed on an object for longer than half a minute. Owing to the great rapidity of the movements, cinematography was of no assistance in attempts at further

analysis. The fundi and visual fields were normal. The performance of the usual tests for cochlear and vestibular functions had no effect on the "convergence tremor." Vujić insists that the movement cannot be interpreted as a nystagmus, and he believes that the cause for this most unusual phenomenon must be sought in a supranuclear localization (convergence center?).

KESCHNER, New York.

THE BASAL METABOLISM OF SOME ORIENTALS. G. MACLEOD, E. E. CRAFTS and F. G. BENEDICT, Am. J. Physiol. **73**:449 (July) 1925.

Careful metabolism studies were carried out on seven Chinese and two Japanese women ranging in age from 21 to 29 years. All of the subjects were normal physically as far as could be determined and had been living under typical American environmental and food conditions for periods varying from fifteen to fifty-two months. The basal metabolism rate was found to be uniformly and consistently low in all cases, averaging 10 per cent less than the commonly accepted standards for American women and 17 per cent lower than the accepted standards for American men. In keeping with these findings, the vital capacity was also found to be definitely below the expectancy for American subjects. Thus the average vital capacity for the authors series was 14.3 cc. per centimeter of height and 1.54 liters per square meter of surface area as compared to values of 20 cc. and 2 liters determined by West (*Arch. Int. Med.* **25**:306 [March] 1920) as representing the average expectancy for American women. Likewise, of a certain significance in this connection was the fact that in the oriental woman the average pulse rate was found to be lower than that generally expected of occidental women; namely, 60 beats per minute with minimum and maximum rates of 54 and 64 respectively. The authors feel that their results, which are in accord with extensive Japanese work, indicate the occurrence in the Chinese and Japanese of a special type of physiology characterized by a comparatively low rate of cellular metabolism. This work is of interest and bears importantly on the general questions of racial physiology and constitution. Practically, too, it is of significance indicating need for especial care in the evaluation of basal rates determined on orientals from the standpoint of clinical diagnosis.

RAPHAEL, Ann Arbor, Mich.

SYPHILITIC ALOPECIA, ITS RELATION TO NEUROSYPHILIS. UDO J. WILE and GEORGE H. BELOTE, Arch. Dermat. & Syph. **13**:495 (April) 1926.

Alopecia as a symptom of syphilis was first described by Fracaster. The relation of syphilis to alopecia has been much discussed; by some the loss of hair is thought to be due to pressure atrophy resulting from a perifollicular syphilitic infiltrate; by others to be due to remote toxins from *Spirochaeta pallida*. In 1914, Cyranka reported a series of cases from which he concluded that syphilitic alopecia is an indication of meningeal irritation.

In the author's series two types are described—thirty cases of essential alopecia, without visible manifestations on the scalp, and seven cases with various kinds of papular lesions. The essential type showed a high incidence of meningeal irritation as evidenced by spinal fluid findings. In one case with negative fluid, there were other symptoms of neurosyphilis. Microscopic examination showed that the essential type was not due to a local pathologic disturbance and was therefore not a true syphilid, while the symptomatic type did show local pathologic changes and was a true syphilid.

In the essential type it was found that loss of hair elsewhere on the body was as marked as loss of hair on the scalp. The authors offer the hypothesis that the essential type is due to a disturbance of the autonomic nervous system, especially in the manner in which it affects the endocrine system. In support of this theory it is shown that loss of hair often results from endocrine dysfunction due to other causes. In the foregoing series relatively more cases of the essential type were associated with leukoderma colli than those of the symptomatic type.

WAGGONER, Philadelphia.

THE RÔLE OF THE HYPOPHYSIS IN INFANTILE DYSTROPHIES. P. LEREBOULLET, Ann. de méd. **18**:452, 1925.

This is a critical review of recent literature. The so-called hypophysis syndromes in children are often produced by lesions of hypothalamic nuclei. Only gigantism and acromegaly may now be considered as pituitary diseases.

DIABETES INSIPIDUS OF SYPHILITIC ORIGIN. J. BABONNEIX and J. LHERMITTE, Ann. de méd. **18**:471, 1925.

This is a report of a case of polydypsia and polyuria. The necropsy revealed a localized leptomeningitis in the region of the optic chiasm and the tuber cinereum. There were perivascular infiltrations and acute hemorrhages in the infundibulum and the tuber region. The paraventricular and supra-optic nuclei were normal, but the cells of the tuber nuclei were reduced in number and showed degenerative signs when stained with Nissl's method.

TREATMENT OF DISEASES, PARTICULARLY OF TUMORS, OF THE INFUNDIBULO-HYPOPHYSIAL REGION. GABRIELLE LEVY, Ann. de méd. **18**:479, 1925.

Antisyphilitic treatment is indicated in cases of syphilitic meningitis producing diabetes insipidus. Radiotherapy is irregular in its results, but is indicated in acromegaly, in which many good results were obtained in cases that were under observation for from three to eight years after the treatment. The surgical method is indicated in cysts of Rathke's pouch and for decompression. Its disadvantage compared with radiotherapy is the impossibility of removing the tumor completely and the high death rate, though this has now been reduced to 30 or 40 per cent by improvement in technic.

WEIL, New York.

FACTORS INFLUENCING THE KNEE-JERK. W. W. TUTTLE, Am. J. Physiol. **72**:50 (March) 1925.

In this communication, Tuttle reports studies made on a series of six apparently normal subjects relative to the influence on the patellar reflex of the following factors: (1) force of stimulus, (2) point of stimulus application and (3) "sleep" phenomena.

With reference to the first factor, it appears that up to a certain point the magnitude of excursion (kymographically recorded) increases with the stimulus strength although seemingly in no definite mathematical relation. At this point, which is reached with forces ranging from about 55 to 110 Gm., depending on the subject utilized, the response becomes maximal and does not

increase with further augmentation of stimulus magnitude. The threshold value, forces below which are ineffective in inducing contraction, also varies considerably with different subjects studied, representing for the author's series a force range of from 14 to 64 Gm. As to the influence of the point of application of the stimulus, it was found that, in general, greater excursions were noted when the stimulus was applied to the midpoint of the patella, to its inferior margin and to the center of the patellar ligament. Relative to the third factor investigated, it was determined that in one subject who reported that his leg and thigh had gone to "sleep," the patellar response became markedly depressed. This reaction resembles strikingly the tendency noted by the author in earlier work in the normal sleep state.

RAPHAEL, Ann Arbor, Mich.

DEMENTIA PRAECOX IN IDENTICAL TWINS. G. H. PARKER, PH.D., J. Nerv. & Ment. Dis. **63**:135 (Feb.) 1926.

The author reports a case of two female twins who had been inmates of the McLean Hospital with a condition diagnosed as dementia praecox. Both had had diphtheria at the age of 16. At the age of 20 both were admitted to hospital with acute excitement, one after a gradual neurasthenic onset, the other with quite sudden onset. For thirty-three years they were patients in separate hospitals. In the protracted phase of their psychosis both talked incoherently, muttered to themselves, showed no power of attention or interest in anything and were subject to impulsive outbursts of noise and destructiveness. After their long separation they met at the McLean Hospital, and there showed striking similarity in behavior and reaction picture. The author declares that there are two kinds of twins, those from a single ovum and placenta — these he calls identical twins and says they are always of the same sex; and those from different ova, protected with separate fetal membranes — these may be of the same or opposite sex. Leeman discovered seventeen recorded instances of dementia praecox in twins; study of these cases helps to distinguish between traits due to germinal inheritance and those due to environmental influence. The simultaneous occurrence of dementia praecox is strongly in favor of a germinal source for this disease.

HART, Philadelphia.

CORRELATED ANATOMICAL AND PHYSIOLOGICAL STUDIES OF THE GROWTH OF THE NERVOUS SYSTEM OF AMPHIBIA. V. THE GROWTH OF THE PATTERN OF THE MOTOR MECHANISM OF AMBLYSTOMA PUNCTATUM. G. E. COGHILL, J. Comp. Neurol. **40**:47 (Feb.) 1926.

Continuing his studies of the growth of the behavior pattern and its mechanism, in this contribution Coghill presents charts accurately plotted to scale of the distribution of the motor nerve cells of the brain and spinal cord of *Amblystoma* at four functionally defined stages in the development of the swimming reaction. These cells are arranged in four longitudinal series on each side distinguished as visceral, tegmental somatic, primary motor and floor-plate cells. These series are separately plotted at each stage and their relations discussed.

The primary motor system differentiates progressively with the scope and extent of muscular contraction and develops very rapidly near the isthmus as the embryo acquires locomotion. In the entire motor column, in the tegmental system and in the visceral system there is bilateral symmetry of

pattern throughout the period studied. The visceral and somatic systems are obviously differentiating under a common factor of control, which appears to be inherent in the nervous system. The major features of the pattern appear to be anticipated in the earliest stages of differentiation within the medullary plate.

HERRICK, Chicago.

THE DYNAMICS OF THE RELATION OF PHYSICIAN AND PATIENT. WILLIAM A. WHITE, Ment. Hyg. **10**:1 (Jan.) 1926.

Man's desire for life called into being the doctor. This desire to remain alive, to get well when sick, is an invaluable asset to the doctor who can mobilize this force in the service of health and assist it in overcoming the destructive forces mobilized by illness. To be able to do this effectively, the physician should not be ignorant of the dynamics of the situation. It is essential, therefore, that the doctor acquaint himself with the personality make-up of the patient; he should know both his assets and his liabilities in the sense of tendencies that make for life or for death; he should be able to evaluate the mental aspects of the situation during illness. Quite unconsciously there exists between physician and patient a parent-child relationship. And it is in the dynamics of the transfer that an explanation can be found of the "cures" that from time to time have been attributed to every conceivable agency. The psychologic component of illness needs recognition and study in order that we may have at our command added ways of mobilizing the forces that make for health.

SCHUMACHER, Philadelphia.

THE EFFECT OF TEMPERATURE UPON THE TONUS OF SKELETAL MUSCLE. F. E. EMERY, Am. J. Physiol. **73**:296 (July) 1925.

In this paper report is made of the influence of temperature on skeletal muscle tonus as determined through study of the knee jerk response in a series of trained subjects. This reflex the author feels may be regarded as an accurate index of muscle tonus. After a series of responses were obtained at room temperature, a five to ten minute record was made at a low temperature (from 54 to 56 F.), following which the temperature was raised to from 70 to 80 F., the recording of the knee jerks being continued for another five or ten minute interval. The effect of drafts of air was also noted. From the data accumulated, Emery concludes that a lowering of temperature is followed by an increase in muscle tonus as measured by knee jerk response, whereas the opposite effect is found to occur when the temperature is raised, the most abrupt change manifesting itself during the first four to eight degrees of change. It was found also that an air draft playing on the subject exerts a reinforcing effect on the tonus change produced by temperature alteration.

RAPHAEL, Ann Arbor, Mich.

THE RELATIONS BETWEEN CENTRAL AND PERIPHERAL COORDINATION. PAUL WEISS, J. Comp. Neurol. **40**:241 (Feb.) 1926.

Various transplantations of limbs were made on larval salamanders by Dr. Weiss, which have been reported in detail elsewhere. These operations were performed after the innervation of the limbs was effected. Later Detwiler made similar transplantations at much earlier stages, before the limb buds received their innervation. The two series of operations give in general similar

results. If a limb is transplanted into the neighborhood of another limb and if it receives its innervation from the limb level of the spinal cord, every muscle of the transplanted limb will, whenever active movement takes place, function simultaneously with the homologous muscle of the nearby normal limb and with the same degree of intensity. Theoretic explanations of this remarkable result have been given by both Weiss and Detwiler. The purpose of this brief paper is to correct some faulty references to Weiss' work; it also serves to introduce to American readers an important research and an interesting theory of central coordination hitherto published only in German.

HERRICK, Chicago.

A STUDY OF PRECIPITATING FACTORS IN INVOLUTION MELANCHOLIA. MARY F. BREW, State Hosp. Quart. **10**:422 (May) 1925.

Histories of twenty-five women and twenty-five men suffering with involution melancholia were reviewed to determine the precipitating factors of the psychosis. The group fell easily into two personality types; about one-half showed a schizoid tendency and the other half evidence of marked inelasticity. The majority showed no somatic disorder of any consequence. Of the women, eight were at the menopause, fourteen had passed it from two to nineteen years before and three were still menstruating.

Of the entire group 42 per cent were assigned psychic causes as the precipitating factor, 38 per cent purely somatic and 10 per cent somatopsychic. The author finds that the same precipitating factors in about the same proportions holds true in other functional psychoses and even in some organic ones. She believes that when the constitutionally predisposed person escapes a psychosis in earlier life he is apt to develop an agitated depression during the period of regression, particularly under physical or mental stress.

HOWARD, Milwaukee.

TOUCH AS A SUBSTITUTE IN THE INTERPRETATION OF HEARING AND CONTROLLED SPEECH. R. H. GAULT, Arch. Otolaryng. **3**:121 (Feb.) 1926.

An interesting account is given of experiments to determine whether the sense of touch can be trained sufficiently to interpret sound vibrations in terms of speech. The author devised an apparatus to conduct vibrations of sound to the skin. He then conducted experiments on himself and on deaf mutes. At first, simple vibrations were used; later, words and sentences. Finally he connected a story of fifty words to which on repetition additional words were, from time to time, added. Suitable tests were made to prove that neither air conduction nor bone conduction to the ears was a factor. The author had in view two objects: first, to provide an additional means by which deaf mutes could understand spoken language; second, by training the skin to perceive vibrations, to have deaf people learn how to modulate their voices. These studies have met with a degree of success that has encouraged the author to continue his work.

HUNTER, Philadelphia

ATYPICAL BULBAR PARALYSIS SIMULATING ANEURYSM. HAROLD W. DANA, Am. J. Syph. **9**:526 (July) 1925.

The patient, a man, aged 44, complained of marked dysphagia and partial aphonia. One year before these symptoms began he had had a sudden attack

of anesthesia of the left side of the body. He now has a spastic left hemiplegia with foot drop and weakness of one hand. He also complains of persistent headache for the last six months, with dizziness and unsteadiness in walking. The admission diagnosis of aneurysm was based on the combination of aortic regurgitation, aortic stenosis and dilatation of the aortic arch with partial aphonia and dysphagia. Roentgen-ray evidence, however, ruled out the possibility of the symptoms and signs being due to pressure within the thorax. Mediastinal pressure, in any event, could not have accounted for the hemiplegia. It would seem most probable that there was a unilateral and somewhat atypical lesion of the medulla localized above the nucleus of the hypoglossal nerve, since this nerve was not affected at all.

SCHUMACHER, Philadelphia.

SUBDURAL AND EPIDURAL HEMORRHAGE, SECONDARILY FATAL FROM HEMORRHAGE OF THE LATERAL SINUS FOLLOWING SLIGHT TRAUMATISM OF THE NECK. HENRI ROGER and ALBERT CREMIEUX, Rev. d'oto-neuro-ocul. **2**:97 (Feb.) 1924.

This article is a full report with complete necropsy findings, of a healthy woman, aged 41, with no hemorrhagic history and no arteriosclerosis, whose occipital bone was fractured and the lateral sinus lacerated by a light blow on the back of the neck. The consequent meningeal hemorrhage produced moderate signs of pressure and headache from which she was recovering; when, on the eleventh day, in consequence of a slight effort in laughing, hemorrhage recurred with death, preceded by coma, three days later. There was no external wound or contusion. The necropsy showed: fissure of the occipital bone on the right passing perpendicularly through the lateral sinus (the bone was deeply grooved by the sinus); hematoma of the right cerebellar fossa at the level of the sinus.

DENNIS, Colorado Springs, Colo.

COMPRESSION MYELITIS SECONDARY TO ECHINOCOCCUS DISEASE OF VERTEBRAE AND KIDNEY. LAURENCE E. HINES, Arch. Path. & Lab. Med. **1**:180 (Feb.) 1926.

The author comments on the rarity of echinococcus disease as a cause of transverse myelitis and cites the literature. A case is reported of a woman, aged 38, whose initial symptoms were shooting pains in the right leg. Numbness and weakness of both legs quickly followed and two months later, at the time of admission to the hospital, typical signs of transverse myelitis were found. Death occurred eleven days after admission. Necropsy findings included compression myelitis of the lower dorsal and upper lumbar cord, localized echinococcus pachymeningitis externa of the cord, multiple echinococcus cysts of the eleventh and twelfth dorsal and first lumbar vertebrae, and a large echinococcus cyst of the right kidney. The author concludes that the kidney was probably the primary seat of infection.

KUBITSCHECK, Philadelphia.

RECKLINGHAUSEN'S DISEASE. HENRY SCHNEIDERMAN, Arch. Dermat. & Syph. **12**:483 (Oct.) 1925.

Four cases are presented showing neurofibromas associated with a varied symptomatology. Two patients were mother and son, and lend evidence to the author's contention that heredity is a factor in all such cases. While the

usual psychic manifestations of feeblemindedness, dulness and apathy were not present, these patients did show marked irritability and undue fatigability. It is believed that this disease is in the nature of an embryonic disturbance of the ectoderm, physical, chemical or nutritional, and that such anomalies are transmitted according to the mendelian law. Since it affects the entire nervous system, as well as the skin, there is no reason to regard occasional tumor formation in the endocrine glands as anything more than a natural concomitant of the disease.

ANDERSON, Philadelphia.

VARICELLA MYELITIS. KNUD H. KRABBE, Brain **48**:535 (Dec.) 1925.

Attention is called to the rarity of this form of myelitis as compared to the frequency of chickenpox. Four cases of varicella myelitis are reported from the literature with as many of polyneuritis which in the author's opinion may have been myelitis. The author's case was that of a boy, aged 8, who developed pains and marked weakness in the lower extremities a few days after his varicella rash. Examination revealed marked paresis of the lower limbs, hypesthesia, analgesia, absent reflexes and bilateral Babinski sign; the spinal fluid was negative. Gradual improvement followed, and the patient was completely cured two months after the onset of the disease. While admitting a possible secondary infection as cause of the myelitis, the author states that this is improbable and that the favorable course of the disease agrees with the good prognosis of chickenpox.

STACK, Milwaukee.

A FURTHER NOTE ON THE MOTOR INNERVATION OF THE DIAPHRAGM. K. SCHLAEPPER, Anat. Record **32**:143 (Feb.) 1926.

"The motor innervation of the diaphragm in mammals is still under discussion." Some claim that the phrenic is the only motor nerve to the diaphragm while others believe the lowest intercostal nerves supply some motor fibers for the portions attached to the lower ribs. Indeed some investigators claim a trophic influence of the sympathetic fibers on the diaphragm and demonstrate a muscular atrophy following section of the sympathetic nerve. The author reported distinct early atrophy of the entire paralyzed side of the diaphragm four months after section of the phrenic nerve in a dog. In the case reported it was found that atrophy was complete after two years.

ALPERS, Philadelphia.

NONBIFURCATING NERVE FIBERS OF THE TRIGEMINAL NERVE. WILLIAM F. WINDLE, J. Comp. Neurol. **40**:229 (Feb.) 1926.

Evidence has been presented by several recent authors that the chief sensory nucleus of the trigeminus is the receptive center for touch and the spinal nucleus for pain. Most of the sensory root fibers of the trigeminus bifurcate on entering the brain, sending one branch to the chief nucleus and one branch to the spinal nucleus. If all of these fibers so divide, the mechanism of central analysis of tactile and painful sensibility presents an interesting problem. Windle finds that in the mouse and pig not all of these fibers divide, but a significant number pass from superficial origin directly downward to the spinal nucleus.

HERRICK, Chicago.

THE FUNCTIONAL RELATIONSHIP BETWEEN THE PITUITARY BODY AND THE TUBER CENTERS. RÉMY COLLIN, Ann. de méd. **18**:428, 1925.

The connection between the hypophysis and the tuber cinereum is not only nervous — myelinated fibers running within the infundibulum — but also hormonic. Collin has shown that the colloid of the middle lobe of the hypophysis can also be demonstrated in the infundibulum and in the perivascular spaces of the tuber cinereum. He emphasizes that the vegetative centers of this region are under the regulating influence of the hypophysis hormone and do not act as independently as recent publications suggest. In his opinion the overestimation of these vegetative centers without taking into consideration their correlation with the hypophysis will prove as one-sided as was the former endocrinologic point of view.

WEIL, New York.

INTRAVENOUS INJECTIONS OF BISMUTH IN GENERAL PARALYSIS. M. DUCHATEAU and P. VERSTRAETEN, J. de Neurol. u. Psychiat. **9**:567 (Sept.) 1925.

The authors report very briefly two cases of general paralysis in women in whom death occurred after intravenous treatment with bismuth tartrate. They consider the cases worthy of publication because, in France particularly, bismuth is used extensively as an after-treatment to malarial inoculations in general paralysis. Acute intoxication with bismuth manifests itself by a primary stage of motor excitement, followed shortly after by paralysis of respiration and cardiac action. Both patients were treated intensively with arsenic, mercury and bismuth.

ALPERS, Philadelphia.

THYROID OPOTHERAPY IN DYSPITUITARISM. JAMES WYNN, J. A. M. A. **86**:820 (March 20) 1926.

After a case presentation the author summarizes as follows: (1) the effect of thyroid extract on a case of dyspituitarism with excessive drowsiness was studied for a period of twenty-eight months; (2) during the first year the patient as a result of imperfect cooperation unwittingly afforded an excellent demonstration of the effects of both insufficient and excessive thyroid administration; (3) on an optional allowance of thyroid he has remained symptom free for thirteen months.

CHAMBERS, Syracuse, N. Y.

THE REGENERATION OF THE GUSTATORY APPARATUS IN THE RAT. BEATRICE WHITESIDE, J. Comp. Neurol. **40**:33 (Feb.) 1926.

The papillae were destroyed by electric cautery, the lesion extending down into the muscular tissue of the tongue. In adult white rats, vallate and foliate papillae regenerate after total extirpation. New taste buds develop in the regenerated papillae and these are formed in the same way as in embryologic development by differentiation of the epithelial cells.

HERRICK, Chicago.

POSTMORTEM FINDINGS IN THE SPINAL CORD IN TWO CASES OF CHRONIC PEMPHIGUS. WALTER M. KRAUS, Arch. Dermat. & Syph. **10**:424 (Oct.) 1924.

Kraus reports briefly two cases of chronic pemphigus, with a fatal termination, in which he studied the spinal cord at the cervical, thoracic and lumbar levels by the methods of Marchi, Weigert and Mallory. He found no pathologic changes in the nerve cells, connective tissue cells, blood vessels, nerve roots or meninges.

PEARSON, Philadelphia.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, March 18, 1926

JOHN FAVILL, M.D., *Vice President, in the Chair*

A PROBLEM IN CEREBRAL DOMINANCE. DR. SAMUEL ORTON.

This article has been published in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY.

MALARIA TREATMENT OF "INSTITUTION" PARESIS; REPORT OF FIFTY-NINE CASES. DR. CHARLES F. READ.

Treatment of general paralysis with malarial infection is the logical outcome of countless observations on improvement following intercurrent disease. The modus operandi is unknown despite many speculations. Favorable results have been reported in cases with few paroxysms and in those with low temperatures so these factors are, quantitatively at least, not important in all cases. Leukocytosis is evidently not essential, though it seemed to be in the older "fever" cures. Formation of specific antibodies has not been proved, nor the suggested change in vascular tonus with consequent hyperemia, transudation and removal of waste products.

Results thus far, as reported, closely parallel those reported in the case of tryparsamide, from 30 to 50 per cent of remissions. Time only will determine the dependability and relative stability of the alleged remissions in the two treatment groups. Reporters should keep in touch with their material and make further reports on the permanence of results after from three to five years.

All patients in the present series were males committed to the Elgin and Jacksonville state hospitals. Diagnosis was on symptoms and serology, plus a careful checkup just prior to treatment in most cases. Inoculation was intramuscular for the most part and 25 per cent failed to take. The paroxysms often developed irregularly and then straightened out into regular tertian seizures, occasionally quotidian. The temperature practically always ran high when the attacks developed typically. The patients sometimes became confused during the fever but, on the whole, took their illness quietly. One case of hallucinosis developed and cleared up.

The well selected cases presented few complications, but three far-gone patients developed a low irregular temperature and died of exhaustion in from three to seven weeks. One working patient died because he was lost sight of before he had had several chills and was much exhausted. Convalescence was prompt and mental improvement began with physical gain in those patients who improved. One case went into remission after the sixth paroxysm. Many cases in this series are too recently treated to pass on as to the final result.

Serologic changes occurred but, as with tryparsamide, did not parallel mental condition. A few spinal Wassermann reactions were slightly reduced; none went negative to 1 cc. The blood frequently became negative. The cells and globulin were reduced, as in practically all forms of treatment. The colloidal gold curve was seldom altered.

The remissions in this group of fifty-nine numbered eleven (18.6 per cent). The average age was 35 in this subgroup and all patients were admitted in 1925. All save two had from twelve to twenty-four chills and the temperature ranged from 104 to 107 F. The mental state was passed on in the Elgin cases by three men with considerable experience, Dr. Nerancy of the Jacksonville Hospital, Dr. Tucker of Elgin (coauthor in this report) and myself; and in the eighteen Jacksonville cases by Dr. Nerancy alone. Every effort was made to arrive at an impartial decision and for this reason two cases are reported with a question, but, on the other hand, several were placed in the much improved group that might very well have been passed as remissions by others, we felt. Previous treatment in the remission group was slight, so far as known. The mental condition before treatment was only poor to fair in all but one of the patients. All developed excellent insight — in fact this was considered a sine qua non.

The marked improvements numbered only seven (11.8 per cent). The average age was 37.6 years on admission; otherwise, there was no marked difference between this group and that of the remissions.

Thirteen patients (22 per cent) were somewhat improved, the majority from what was considered poor condition before treatment to fair afterward. This change, while of not much benefit to the patient in the long run, means easier care and a more comfortable status, less risk of injury, greater cleanliness, etc. The average age on admission was 41 years and all but four patients were admitted in 1925. What may be significant in this group is the fact that only four patients had ten or more paroxysms.

Stationary cases numbered twenty-one (35.6 per cent). The average age on admission was 40 years and many patients had been in the hospital two or three years. In nine cases there had been less than ten paroxysms and five were cases in which arrest had been secured with tryparsamide. The failure to improve might well be attributed to irreparable brain damage.

Three cases failed mentally and the four patients who died have already been mentioned. Four cases already in apparent remission and not included in this series were given a full "course" without mental or physical damage resulting.

A year ago we reported results in forty-two cases of general paralysis treated with tryparsamide during 1923 and 1924 at Elgin and it is interesting to note that the percentage of remissions obtained (19 per cent) was practically the same as reported with the present group. The improvements were about the same in number and many of these patients are still living, not a few on parole of the grounds, who, statistically speaking, should have been dead a year or more ago. Several of the remission patients have died.

Malarial treatment is much easier than weekly intravenous injections. Whether the patients will "hold up" as well remains to be seen. Our present cases must be followed up carefully.

Regular Meeting, April 15, 1926

SANGER BROWN, M.D., *President, in the Chair*

INVOLVEMENT OF THE CENTRAL NERVOUS SYSTEM SECONDARY TO PRIMARY CARCINOMA OF THE LUNG. DR. HARRY L. PARKER (by invitation).

This article will be published in full in a later issue.

THE DIAGNOSIS OF PSEUDO-OPTIC NEURITIS. DR. VIRGIL WESCOTT.

Our ideas as to the picture presented by the inflammatory and edematous conditions which may be studied in the intra-ocular portion of the optic nerve are certainly poorly defined and a multiplicity of names has added to the confusion. We should first distinguish between a true neuritis and a true edema not by stating arbitrarily that a neuritis represents a swelling of 2 diopters or less while an edematous nerve head has a swelling of 2 diopters or more. In the so-called descending neuritis the swelling and hyperemia are confined to the disk and immediately surrounding retina without overfilling of the retinal veins. In neuroretinitis the nerve head is hyperemic, the margins of the disk are blurred, and the retina is edematous, hemorrhages being scattered out toward the periphery of the fundus. In choked disk the lesions are confined to the nerve head with hemorrhages on or near the swollen nerve and with distention of the retinal veins. Risley pointed out long ago the hyperemia of the disk and retina associated with other symptoms of eye strain which disappeared with prolonged use of cycloplegia and remained in abeyance on the wearing of the proper correction for the error of refraction and with due attention to matters of ocular hygiene. Likewise, certain low grade chronic infections or toxemias not alone from focal infections but also from alcohol, tobacco and gastro-intestinal disorders cause changes in the disk characterized by hyperemia and even blurring of the margins associated with fading central vision and central scotomas, especially for red test objects. In arteriosclerosis of the fundus oculi there is at certain stages a dull red congestion or even an actual edema of the optic disk.

In using an ophthalmoscope routinely one often notes disks which are fiery red, the surface being covered by an edematous layer, the margins blurred and veiled by striations composed of fine grayish lines and minute capillaries, with no central depression or cup and the general impression of protuberance. To this nerve picture may be added tortuosity and fulness of the retinal vessels. The perivascular lymph sheaths are unduly prominent in the form of white lines along the central vessels, especially the veins. If associated with headaches, nausea and vomiting such an appearance arouses suspicion and calls for further investigation. To such a picture the ophthalmologist gives the name pseudo-optic neuritis. The neurologist is prone to designate such an appearance as that of a low grade or beginning neuritis. Such papillae are sometimes found in perfectly normal eyes, but more frequently in eyes affected with marked hyperopia, astigmatism or congenital amblyopia. They are often found in conjunction with irregularity in the shape of the papilla, an abnormal division of the vessels or a crescent at the border of the disk and are to be thought of as an abnormal formation of congenital origin. The eye develops an evagination of the medullary canal, this evagination being transformed into the primary optic vesicle, the latter being separated from the medullary canal by the pedicle of the optic vesicle. This pedicle becomes the optic nerve as the nerve fibers of the retina push in from the secondary optic vesicle. The aperture in the sclera at the posterior pole of the eye through which

the nerve passes is traversed by a transverse network of connective tissue fibers containing much elastic tissue—the lamina cribrosa. The fibers of the nerve fiber layer of the retina pass through the meshes of the lamina cribrosa and on the posterior side become surrounded by medullary sheaths. In the small farsighted or hyperopic eye the optic nerve enters through a small opening in the sclera and choroid, crowding together the nerve fibers, obliterating the optic cup and concealing the lamina cribrosa. In such an eye the retinal vessels are tortuous and often apparently distended. The imperfect coaptation of the invaginated walls of the secondary optic vesicle gives to the retina a shot silk appearance with its conflicting reflexes.

The diagnosis of an optic nerve lesion cannot be made solely on its appearance as viewed with an ophthalmoscope, nor can it be made always on a single examination, for the appearance of any pathologic nerve may change from time to time. Beard observed a case for twenty years in which there was 9 diopters of swelling without enlargement of the blind spot, but in which the patient suffered from headaches and vertigo, relieved by the correction of the hyperopia with gradual improvement in the vision, which had been very poor at the first examination, but without material change in the appearance of the nerve head. The vision with and without the proper correction must be taken. An opportunity to determine the error of refraction under cycloplegia should be part of every neuro-ophthalmologic investigation. The mydriasis gives greater opportunity for study of the eyegrounds and, as de Schweinitz points out, "we shall often be misled as to the progress of disease and its effect on the sight if, as is too often done, we test the visual acuity without attempting to correct as far as we can any existing error of refraction." The neurologist is always interested in field studies which should be made under ideal conditions, with the proper equipment, and repeated frequently to note the progress of the lesion and to educate the patient in the matter of exactness of observation. While changes in the periphery of the field are important, equally so are studies of the blind spot and paracentral area. Observations made roughly or only in the principal meridians do not uncover defects noted with various sized and colored test objects carried on the arc at ten degree intervals.

When a patient is referred to a neurologist because of symptoms referable to intracranial disease, whether the result of tumor or inflammation, ophthalmoscopy may be an aid in determining the presence of increased intracranial pressure. On a pseudo-optic neuritis diagnosed as such at the first examination may be superimposed later a real neuritis or a choked disk noted as such at a subsequent examination. Such a sequence of events has led many neurologists to doubt the accuracy of the original diagnosis, but such a situation need not be a matter of chagrin to the ophthalmologist or of surprise to the neurologist. Even in true choked disk the amount of edema varies from time to time giving rise to the misconception in those cases in which the choking subsides following some extracranial operation that the removal of the focal infection demonstrates that the latter was the cause of the papilledema. Pseudo-optic neuritis is a distinct entity and hundreds of cases have been observed by ophthalmologists over a sufficient length of time to establish it as one of the nerve head pictures not associated with inflammation or edema of the optic nerve secondary to intracranial disease.

DISCUSSION

DR. HARRY GRADLE: I think Dr. Wescott's remarks are particularly apropos in that there still seems to exist in the minds of many a great confusion as to the main conditions in the optic nerve head. The pseudo-optic neuritis which has

been so well described must be remembered so that we do not fall into the error of calling it a pathologic condition. An edema or papilledema of the optic nerve head is primarily not an inflammatory condition. It is an edema purely and simply from increased cerebrospinal pressure within the sheaths of the optic nerve. When such pressure has been in existence a greater or lesser length of time a secondary neuritis occurs, and it is due to this that we find hemorrhage and exudates. A pure papilledema has no hemorrhage and no exudate.

A true papilledema must be diagnosed in three ways. We must have the visual field observations, the neurologic interpretations and the opthalmologic picture. If we do not have these three things we are not justified in calling the condition a papilledema. On the other hand, an optic neuritis is inflammatory, purely and simply. It is a real toxemia and is not due to edema or pressure. It is a mistake to call the two by the same name. They are different clinical entities and of different etiologic import. Only too frequently do we hear a choked disk called papillitis, and vice versa.

Dr. Hugh T. Patrick: Something like forty years ago I was introduced to the pseudo-choked disk of adolescence and for the last forty years I have tried to find some explanation for it. At long intervals I have seen the condition. I have spoken of this to several opthalmologists but have found no one who could explain it.

Dr. Wescott: I have not seen one of the cases that Dr. Patrick describes, but the cases of pseudo-neuritis I have seen, and have seen the cases so designated many years ago. They do not change from year to year. In all of them the eyes are small, hyperopic, with the nerves crowded together, and all the vessels tortuous; and no explanation that I know of has ever been satisfactory, any more than the case which Beard watched for so many years with the swollen nerve head, the difference in the disks being anywhere from 4 to 6 diopters.

The diagnosis of any optic nerve lesion must be made not by simply looking at it with an ophthalmoscope, and not by noting the margins or appearance of the cupping, or an absence of a clear view of the lamina cribrosa, because we see every day nerves of different size and shape, the vessels situated at different levels, divided at different levels. The diagnosis must be made on the ophthalmoscopic, the field changes, and the general neurologic examination, as Dr. Gradle has said.

Cerebral Repair After Injury. Dr. Wilder Penfield.

The study of brain wounds was carried out in part by me alone and partly in conjunction with del Rio-Hortega.

Normal interstitial cells are divided into neuroglia and microglia (or third element). The term neuroglia covers (1) fibrous and protoplasmic astrocytes or classic neuroglia, and (2) the numerous group cells, called by del Rio-Hortega oligodendroglia. Microglia (third element of del Rio-Hortega) differs from neuroglia both in behavior under pathologic conditions and in origin.

It was found that nerve cells and oligodendroglia did not play any rôle in the scar formation.

Compound granular corpuscles could be seen on the borders of the wound by the third day and all transitions from complex microglia to these phagocytic cells were to be seen about the wound. These granular corpuscles were present in the wound as long as products of destruction were present. After that time microglia in its complex spider-like form could be seen in the scar. No evidence was seen of transformation of neuroglia cells into compound granular corpuscles.

Neuroglia astrocytes reacted rapidly also. There was swelling of these cells about the wound within twenty-four hours. At the end of three days these cells

and their expansions were swollen and some cells at the very border of the wound were undergoing ameboid degeneration. By the fourth day multiplication of neuroglia had begun and by the sixth day it was quite marked. The cell division was amitotic. From the sixth day on, the astrocytes came to be arranged in a concentric fashion about the wound with their large expansions radiating like the spokes of a wheel with the wound as the hub.

During the process of swelling glia fibrils disappeared, but soon after cell division had taken place fibrils were laid down again so that the scar was made up of fibrous neuroglia whether in gray or white matter.

Connective tissue cores formed in the stab wounds and new collagen fibers were laid down and plentiful by the nineteenth day. Collagen fibers were seen to be heavy on and about the vessels in the center and borders of the wound.

It was found that some wounds, however, particularly deep in the white matter, did not fill with connective tissue but remained gaping. About these gaping tracts there was less or no gliosis and when the gliosis was present in these cases, the radial arrangement was not. The stab wounds with connective tissue cores showed evidence of contraction while the empty wounds did not.

An adhesion at the surface of the brain was shown in which vessels had evidently been pulled from out the brain, inasmuch as they had carried with them the vascular feet and expansions of neuroglia cells, while the cells whose expansions were being pulled on could be seen at the surface of the brain.

It was suggested that the mechanism of contraction of brain wounds was contraction of the scar tissue in the center which resulted in traction on the various vessels which enter the scar. These vessels in turn pull on the astrocytes whose footplates are wrapped around them.

This progressive contraction might help to explain the marked reduction, in the threshold of the convulsant dose of wormwood oil, produced by a simple incision in the brain of an animal.

In cranioplastic operations one aim should be to prevent vascularization of the brain wound from without, so that vessels in the superficial scar might be prevented from pulling on the vessels in the brain below.

DISCUSSION

DR. G. B. HASSIN: The work presented tonight by Dr. Penfield has been conducted mainly by the method of del Rio-Hortega of the school of Ramón y Cajal. The microglia cells playing, according to Penfield, so great a rôle in the process of brain repair are considered by their discoverer del Rio-Hortega and his followers mesodermal elements. If one studies the classic researches of Jacob on secondary degeneration, one can follow up the successive transformation of the glial elements into complicated structures looked on by Hortega, Penfield, and other able workers as microglia, i.e., connective tissue cells. If such is the case, the entire neuropathology will have to be revised. Thus the degenerative phenomena seen in amyotrophic lateral sclerosis, multiple sclerosis, subacute combined cord degeneration and other conditions will have to be revised for the purpose of ascertaining whether the mesodermal or the ectodermal elements play the preponderant rôle in the brain tissue repair. The new teaching also upsets the fundamentals of Nissl's theory that the mesodermal elements, under normal conditions, cannot be intermingled with the ectodermal ones. One point, however, is definitely settled by the work of Hortega and his followers, that the numerous mononuclear cells scattered throughout the gray and white substances are not hematogenous elements which they resemble so much with ordinary methods of

staining. Other cells scattered in immense amounts over the subcortical white substance and much resembling the microglia are considered by the Hortega school as glia and called oligodendroglia. As I understand, even the Madrid school did not come to any definite conclusion as to the nature of the microglia and oligodendroglia. It will require much work to reconcile their views with that of the school of Alzheimer. So far little has been done along these lines, as comparatively little use is made of the Spanish methods in this country and abroad. This is mainly due to the fact that the Spanish literature is not easily accessible on account of the language, while the abstracts in other foreign languages are even more trying to one unfamiliar with them than the original contributions in Spanish. Thanks to the efforts of Bailey and Hiller, Solomon Fuller and especially the contributions of Penfield, the value of the methods of Hortega and the mode of their application became in this country better known and understood.

DR. PENFIELD: I agree with Dr. Hassin that it is necessary to take a new point of view in neuropathology. By the recognition of microglia and the establishment of the origin of the phagocytic cells from microglia a real step toward simplification has been made. The large number of types of cells which are talked about and described, particularly by the German school, is confusing. During the past year Dr. Cone and I have been trying to correlate the work of the German and the Spanish schools, studying certain pathologic conditions as stained by the different methods. The result of application of the Spanish methods has not been a contradiction to the work of the German school, but it has simplified many complicated pictures. I think the time will come when it will be acknowledged that the macrophages develop from microglia and not from neuroglia, just as they are derived from mesodermal tissue elsewhere in the body. They can be seen in a wound in which there has been a decompression. Above, in the muscle of such a preparation, the macrophages and below, in the brain, the Gitterzellen may be stained by microglia technic in the same section. This puts the process following destruction in the brain on exactly the same basis as that which follows destruction elsewhere. The rôle of neuroglia begins after the "scavengers" have gone to work. The activity of these cells ends in the formation of a scar.

A CASE OF SUPRASELLAR CYST SUCCESSFULLY OPERATED ON. DR. HARRY S. GRADLE (by invitation).

The case presented was that of a suprasellar cyst presenting only ocular symptoms, and those essentially a loss of vision and visual field changes. The operation was done by the temporofrontal route, the anterior wall of the cyst being removed. There was recovery from operation, and recovery of practically half of the normal vision and the majority of the visual fields.

DISCUSSION

DR. GEORGE W. HALL: I have observed a case in a school teacher who manifests some of the things Dr. Gradle has brought out. There is a marked difference between what the patient complains of and what is found objectively so far as the disk itself is concerned. This case occurred in a patient of Dr. Findley's who was inclined to think he was dealing with an hysterical case, because of the contracted fields of vision, although he could find no changes in the optic disk. The sella was normal, but after studying the case a short time Dr. Findley discovered a slight trace of optic atrophy. She was sent to the Mayo Clinic and the physicians there were not sufficiently satisfied to recommend operation. She

was sent home and was studied for a short time longer. The vision became steadily worse, the headaches were more or less persistent, and we felt convinced that there was a growth in the region of the sella. She was returned to the Mayo Clinic and operation was performed by Dr. Adson. He found a suprasellar growth which was quite vascular. He was unable to remove it entirely but the patient's condition improved. At present she is receiving roentgenotherapy. The field of vision has not changed much, but since the operation she has developed some epileptiform attacks. The thing that impressed me was that the disk showed no abnormality whatever for a long time as compared with the narrowed fields of vision.

NOTE: Under treatment the epileptiform attacks have stopped, the headaches are mild and the vision has improved.

DR. GEORGE L. DAVENPORT (by invitation): This case has several interesting phases. You will note that the approach was from the left side because the neighborhood symptoms, complete blindness of the left eye, indicated a greater extension of the lesion to this side. The brain had to be retracted upward, mesially and backward and naturally there was a possibility of an aphasia developing, but fortunately no aphasia or olfactory disturbance developed.

Suprasellar lesions often spring from locations other than the pituitary body, that is, from the anterior perforated area, sheath of the optic nerve or embryonal remains. All suprasellar lesions, even when they are sellar in origin, present symptoms referable to the optic chiasm and it seems that the term "chiasmal lesion" is more descriptive, meaning lesions in and about the chiasm. We believe that the indication for the intracranial approach is indicated (1) in all cases of suprasellar lesions, (2) in cases presenting optic atrophy with the typical yellowish discoloration of the disks so as to save if possible any vestige of vision remaining and (3) in cases in which there is doubt as to whether the lesion is suprasellar or still confined to the sella itself.

Lesions of sellar origin that involve the optic chiasm and do not present a bitemporal hemianopia but are progressive, not only sudden in their onset but also rapidly progressive, are apt to have marked intracranial extension. We must also take into consideration the pituitary dysfunction which is either due to primary involvement of the pituitary body or the result of pressure.

The operation involved the laying down of a large osteoplastic flap of the frontoparietal temporal bone. The cyst extended slightly anterior to the chiasm but mostly posterior and its wall consisted of the thinnest membrane; hence, on opening the cyst the wall retracted markedly in all directions. There was no evidence of solid growth in the cavity and in trying to grasp a portion of the retracted cyst wall for removal, some bleeding started which was controlled. We did not get a portion of the cyst wall for examination and the cyst probably sprang from a craniopharyngeal duct remains. It is now about eighteen months since the operation with no evidence of recurrence up to this time.

DR. D. M. OLKON: In a case recently studied at the Educational and Research Hospitals of the University of Illinois, Chicago, the patient, a girl, aged 15, was sent to our dispensary from a neighboring town by an eye specialist with the report that the patient was complaining of occasional headache and poor vision. In spite of the prescription of glasses, the failing vision continued.

Physical examination showed the girl to be well developed for her age with no bodily signs of disease. Neurologic examination was entirely negative except for definite observations in the visual field and optic nerve changes.

Perimetrically, the field of vision was markedly restricted with a bilateral hemianopia; color perception was very limited. Red-green, blue-yellow and even white were uncertain. There was optic atrophy without a papillitis. The roentgen ray showed a right opaque frontal sinus (the other sinuses normal) and a widened sella turcica with eroded anterior clinoid processes. There was no polyuria and no disturbance in fat metabolism nor were there changes in the sugar values in the blood or spinal fluid. Continued observation showed a progressive shrinkage of the visual field, particularly to the temporal sides.

The question was to determine the pathologic processes present: was it a suprasellar cyst, a hypophysial tumor, a third ventricular neoplasm or a new growth in the chiasma itself. In the absence of all organic neurologic and general somatic manifestations of the entire body, in the presence of the eyeground evidence mentioned above in combination with the definite perimetric observations and in the absence of the papilledema, the diagnosis was inclined toward a rapidly growing neoplasm of the chiasma.

The patient went to Boston to consult with Dr. Cushing who operated and reported to us that on operation he found a hard tumor in the chiasma which he did not entirely remove.

This case is instructive in that the principal observations were disturbances in the visual field, with optic atrophy, but without any other pressure signs and bodily or vasomotor disturbances.

Dr. Wilder Penfield (by invitation): This case of Dr. Gradle's is very interesting. I have recently studied a similar case of suprasellar cyst in which operation was performed by Dr. Elsberg. The tumor was growing above the pituitary itself, but it was made up of the acidophilic cells of the pituitary. We have found in some of these cases that while the patients do well after operation they may develop headaches later. This type is much benefited by high frequency roentgenotherapy, particularly if it is used from both sides. The important thing about the operation approach which Dr. Davenport has used is to make a large incision which goes right up to the midline. If used thus and the ventricles are dilated the transfrontal approach is extremely satisfactory.

Dr. Gradle: During the last year the patient has been entirely free from symptoms. He has had no headache and no ocular symptoms, and during the last six months vision has improved slightly. Extreme distance vision is a little bit difficult, but near vision shows no interference. So far as the other functions of life are concerned he carries on just as he did before the cyst appeared.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, March 18, 1926

Donald Gregg, M.D., *President, in the Chair*

Experiences with Feebleminded Problems. Dr. William Healy.

What is feeblemindedness? It is clear that we can define mental defect according to some arbitrary norm established for certain groups. It must be understood that this is an arbitrary arrangement, as when the intelligence quotient is used, and in any sort of fairness it has to be interpreted differently for different groups because the capacities of different groups vary so much; the results of tests are also affected by educational training to a certain extent. This

is notably true of the ordinary age-level tests in which the requirements are so largely verbal, and the child from the educated family, as shown by certain special researches, more readily passes certain tests, proving that they are not safe indicators of innate ability. Keeping group norms in mind we can state that mental defect may be of various grades and also may exist for various special mental capacities.

Feeblemindedness is a matter for arbitrary definition; it implies comparison of one person with others, and a social situation is thus involved. It seems to me that the general feeling among specialists concerning feeblemindedness is that it would be best to follow, somewhat, the British definition and consider the feebleminded person as one who, on account of his mental defect which has existed from birth or from an early age, is unable to perform his duties or conduct his affairs as a member of society in his particular social group. Let this be true, and we still have confronting us the question, Who is commitably feebleminded? In practice one finds that it is desirable to commit for the following reasons: (*a*) in order to educate as much as possible, sometimes with the idea of later paroling into the community the person who has been institutionally trained; (*b*) for the protection of the person, or (*c*) for the protection of the community from the person.

But all through, whether considering mental defect or feeblemindedness, any good study of defectives leads us to discern the important fact that there are immense differences between them, not only in such matters as auditory memory or visualizing ability, which are of special import for the educationalist, but also in the vocational field, because of the vast differences in language ability, motor skill, mechanical aptitude, musical ability, and, very important, dynamic qualities. I have published cases illustrating the differences in this matter of forcefulness and energy. I have seen a mentally defective boy, for example, on account of his energy able to get a job much easier than almost any normal boy with whom I have come in contact. Indeed, we are soon led, even in this field, to study characterology and what makes differences in personality traits, even in this simpler group of persons. In studying their conduct tendencies, we note great variations in insight, quality of mental content, and ideation. If one undertakes to offer a prognosis concerning mental defectives, one has to take into account all of these factors, as among the normal.

It is clear that in institutions for defectives there is an opportunity for the educational psychologist that has never been utilized. There are splendid chances for making a study of comparative abilities, of the educational possibilities, of special aptitudes and of the background of personality and character traits, all of which might be of the greatest service for developing our knowledge of the ordinary run of people. Often the best opportunities for gaining knowledge are found through studying simpler organisms in a simple environment, and I believe that this might be the case if much deeper researches were made into the mental life of defectives.

INTERIM REPORT ON THE THIRD SERIES OF TEN CASES (WAVERLEY RESEARCHES IN THE PATHOLOGY OF THE FEEBLEMINDED). DR. OSCAR J. RAEDER.

In this series of researches begun by Southard, Fernald, and Taft in 1918 (Mem. Am. Acad. Arts and Sc. **14,** May, 1918, and ibid, **14,** December, 1921), the prime object was to establish a correlation between testable mind and measurable brain and to find preventable causes, disease processes, that formed the basis of retarded and defective mentality. In the second volume, it was shown in a comparison of the mental ages with the pathologic conditions

(histologic and gross) of the first twenty brains studied, that a certain definite correlation existed. The obviously pathologic brains — in cases of arrested development, porencephalic, some microcephalic and most hydrocephalic — showed a lower mental age. The better anatomic specimens at the upper end of the ladder, including more uniform and complex brain patterns, showed consistently higher mental ages. However, there was a wide midzone in which marked exceptions occurred. Most striking among these were two patients, i. e., case 3, a microcephalic patient (brain weight 610 Gm.) with a mental age estimated at 7 years, and case 14, a large imbecile (brain weight 1,205 Gm., and of a fairly complex pattern), who had a mental age of only 1.3 years. This seems to demonstrate either that we are unable with our present histopathologic and microchemical technic to discover the differences in the quality of the nerve cells, or that some biochemical process, similar to that involved when the cretinic dullard is brightened up by thyroid substance, may be responsible for the proper functioning of the ganglion cells. But on account of the relatively small number of carefully studied cases in the literature, it seems imperative that a large number of feebleminded patients with careful and extensive clinico-anatomic observations should be adequately studied and recorded before we can form the foregoing conclusions.

The following is a brief summary of the observations in five of the third series:

Case 22, adult imbecile, mental age 7, brain weight 1,000. Miliary tuberculosis with rare tubercles in the cortex. There was considerable satellitosis and gliosis.

Case 24, adult idiot, mental age estimate 1.2 years, brain weight 1,220. Satellitosis, neuronophagia.

Case 27, Mongolian idiot, mental age 2.1 years, brain weight 1,275. Increased fat deposit in ganglion cells with marked nuclear eccentricity, some heterotopia.

Case 28, imbecile, aged 19, mental age 8.2 years, brain weight 1,385. Many binucleate ganglion cells were found in different areas; also nerve cell sclerosis, pyknosis and architectural dilapidation.

Case 30, cretin, aged 37, mental age 6 months by the Stanford scale, brain weight 1,105. Marked richness of ganglion cells (no aplasia), many sclerotic nerve cells. This case seems to uphold the dysfunction theory for some types of feeblemindedness.

RESEARCHES ON FEEBLEMINDEDNESS. DR. ABRAHAM MYERSON.

This is a summary of a research carried on under a grant from the Research Division of the Massachusetts Commission of Mental Diseases, during the years 1924-1925, and which is in progress at present. The research material was drawn mainly from the population of the Walter E. Fernald School for Feeble-Minded at Waverley, Mass., and the late Dr. Fernald and his associates cooperated in the work. The biochemical work was done by Dr. Louis J. Ullian; the statistical analysis of the figures on heredity was carried through by Dr. Henry B. Elkind. Miss Hannah J. Smith did the field work and assisted Dr. Elkind in the statistical analysis. Dr. Abraham Myerson made physical examinations of the 845 patients involved, and, in general, directed the research.

1. *Definition of Feeblemindedness.*—None is satisfactory, since the concept is a changing one, and the upper limit which distinguishes feebleminded from low-grade normal is, practically speaking, arbitrarily chosen. The classification is in part based on clinical distinctiveness, as the Mongolian idiot, in

part on pathology, as in the case of the cretins and the organic brain cases, another group according to supposed etiology, as in the case of the so-called congenital syphilitic patients. With the exception of a few other scattering groups, such as hydrocephalic and microcephalic cases, the bulk of feeblemindedness is entirely unknown as to etiology, pathology and therapy. Ordinarily, this unclassified group is subdivided into idiot, imbecile and moron, with arbitrary limits separating one group from another. These terms represent in no sense a biologic classification. Cretins, for example, may be either imbeciles or idiots; the same is true of Mongolians; the organic brain cases range through the three groups, and in any family in which heredity is invoked as a cause or essential condition, one may find all three types of the feebleminded. Therefore, these terms, idiot, imbecile and moron, are merely a convenient form of measurement, help in the institutional care and in provision for the future, but give us no information as to cause or pathology.

Our 845 cases have been grouped under the following headings: spastic 82; congenital syphilitic 69; cretin 19; Mongolian 37; microcephalic 17; hydrocephalic 9; unclassified 612. The term "unclassified" represents the cases in which no known pathology, etiology or specific clinical grouping can be ascertained. As will be seen, this group includes a large number of cases in the etiology of which "heredity" plays an important rôle in a relatively large percentage of cases.

2. *Heredity of Feeblemindedness.*—We have limited our study of the heredity of feeblemindedness to the incidence of mental diseases, epilepsy and feeblemindedness in the parents and siblings of the patients involved. We have omitted other pathologic traits simply because there is no way of evaluating them, and because their incidence as psychopathic characters is entirely an assumption. Thus, headache, nervousness, criminality, alcoholism, chorea, paralysis, strabismus and the like polymorphic conditions have not been considered. Furthermore, we have limited ourselves to the parents and siblings because, in the social strata from which most of our patients have been drawn, precise information concerning collaterals and remote ancestors is, practically speaking, valueless. We have felt that we could obtain a certain value for our figures by limiting the field of inquiry. The figures on which the following conclusions have been made will appear in detail in a forthcoming paper by Drs. Elkind and Myerson. We cite here the conclusions of Dr. H. B. Elkind in respect to the statistical analysis.

"In conclusion it may be stated that a statistical analysis of the data presented to me for study indicates for, at least, the Walter E. Fernald State School:

"First, that mental diseases and epilepsy bear no definite relationship to feeblemindedness as to inheritability, except possibly an accidental one; and

"Second, that there appears to be a distinct tendency for the inheritability of feeblemindedness from feebleminded parents alone, the trend being almost an incontrovertible one in the case of descent from two feebleminded parents and quite a distinct trend in the case of descent from a single feebleminded parent; and

"Third, that it is common for one feebleminded sibling to be born to apparently normal parents."

In addition to this, Myerson adds: "In the case of the classified groups, spastics, cretins, Mongolians, etc., with the exception of the syphilitic feebleminded, there appears to be no relationship to inheritance of any kind; and

"Second, that it is in the case of the unclassified group that the inheritance from feebleminded parents appears very prominently; and

"Third, that the syphilitic feebleminded have about the same incidence of feebleminded ancestors as do the unclassified feebleminded. This will be discussed later in relationship to the entire question of syphilis as a cause of feeblemindedness."

It may be stated that feeblemindedness, when inherited, appears to have no relationship to anything else except feeblemindedness; that, therefore, as a biologic problem, feeblemindedness is distinct from the mental diseases and epilepsy.

3. *Clinical Study of the Sporadic Cretins.*—(*a*) We have no new physical features to add to the diagnosis of sporadic cretinism, except that the bridge of the nose of the cretin appears to be definitely lacking in bone, and is very flexible. This is in striking contrast to the condition, for example, found in the Mongolian idiot and in the normal person.

(*b*) We find no evidence that sporadic cretinism is related to heredity, either of endocrinal disorder or in relationship to feeblemindedness, mental disease or epilepsy.

(*c*) The spinal fluid of cretins is entirely negative.

(*d*) We corroborate the observations of other workers that there is a low basal metabolism, and that cretinism is undoubtedly a deficiency disorder of the thyroid gland.

(*e*) The biochemistry of cretins presents the following picture: The sugar tolerance is distinctly erratic, in some cases being nearly normal, in others corresponding to a low sugar tolerance, and in still others to a high sugar tolerance. The carbon dioxide combining power is distinctly lower than in established normals. There is, perhaps, a slightly increased amount of calcium found in the blood. The hematologic picture is frequently that of a moderate secondary anemia.

4. *Clinical Study of Mongolianism.*—(*a*) We have nothing to add to the physical characteristics of the Mongolian.

(*b*) There is no evidence that Mongolianism is in any way related to heredity, either of endocrinal disturbance or in relationship to mental diseases, feeblemindedness or epilepsy.

(*c*) While the Mongolian is frequently the last child in a large family, it is about as frequently the first child or the middle child. As far as we can see, there is no real relationship between the order of birth and the age of the mother, or any other such factor in the incidence of Mongolianism.

(*d*) Recently, Jenô Vas, in the *Jahrbuch für Kinderheilkunde* (**61**:51, 1925), makes the definite statement that Mongolian idiocy directly results from suprarenal insufficiency, and gives the following four signs: (1) a low blood pressure; (2) the vasoconstriction or white line of Sargent; (3) slow coagulating time of blood; (4) nondilatation of pupil after use of epinephrine chloride. Repeating his work, we have not been able to corroborate it in any particular. We do not find evidence that Mongolian idiocy is a disorder of the suprarenal gland.

(*e*) The Mongolian frequently has chronic infection of the ears and nares. It is perhaps in relation to this that one finds a mild secondary anemia common among the Mongolians.

(*f*) The biochemistry of the Mongolians is, on the whole, negative. In a few cases there is a little disturbance in sugar tolerance, which, however, did not appear to us to be significant.

5. *Clinical Study of Spastics or Organic Brain Disease.*—Of these, there was a considerable number at the Waverley School for Feeble-Minded. Our own cases gave us no definite information to add to the knowledge of the subject. We find no evidence that mental diseases, feeblemindedness or epilepsy are related in any hereditary way to these conditions. The recent literature emphasizes the rôle of birth trauma, and we call especial attention to the recent work done by Ylppö, Sietz and, especially, Philip Schwartz in Germany, and to the recently reported observations in the spinal fluid by Monroe, Falls and Sharpe in the United States.

We attempted to study from the records of the Boston Children's Hospital the rôle of encephalitis in early childhood in the etiology of feeblemindedness. We had an insufficient number of cases from which to draw conclusions, and can only state that a large proportion of the thirty cases were followed by alteration in mentality of a type severe enough to be classed as feeblemindedness. It is likely that unsuspected birth trauma and infections of the encephalon occurring early in life account for a good deal of the feeblemindedness in nonhereditary cases. Biochemically, the spastic cases are negative.

6. *Syphilis as a Cause of Feeblemindedness.*—In a previous paper, read before this society, March 19, 1925, we stated that, in our opinion, the statement in the literature that syphilis caused feeblemindedness cannot be substantiated. We may qualify this by saying that if it does cause feeblemindedness, it does so only in a small number of cases. Our reasons for this conclusion are as follows:

First, the amount of syphilis in the schools for the feebleminded does not exceed what might be expected in children drawn from the social stratum from which these patients come.

Second, the physical examination of the congenital syphilitic patients in the institution for the feebleminded does not show any especial clinical picture, except in the cases in which definite neurosyphilis is present, and this is relatively infrequent.

Third, the examination of the spinal fluid is negative in every way, except in a few cases in which definite neurosyphilis is present (congenital general paralysis and congenital tabes).

Fourth, the neuropathologic work done on congenital syphilis in the institutions shows a picture not at all like that found in neurosyphilis in general. Anomalies of brain architecture occur, but these occur also in the feebleminded conditions of nonsyphilitic origin. Except when definite neurosyphilis exists, the picture is not at all characteristic from the standpoint of syphilis.

Fifth, in our sixty-nine cases, there was a striking similarity between the heredity of the syphilitic group and the heredity of the unclassified group; namely, that a large proportion of feeblemindedness occurs in the parents and siblings of these patients, much larger, for example, than occurs in the families of the spastic patients.

The hereditary picture lends weight to the belief that the syphilitic feebleminded cases are not of known etiology, and that the syphilis is merely incidental. It is our belief that syphilis operates on the all or none principle in relationship to the involvement of the brain. Either it is frankly neurosyphilis,

or it has no relationship to whatever mental disease is present. Biochemically, the syphilitic cases are negative.

This concludes the work done up to the present. Details in respect to each subdivision of the subject will appear in separate publications. We state it as our belief that the problem of feeblemindedness is a problem *sui generis*, and that the subject matter of feeblemindedness must be broken up into groups, and each clinical group studied intensively as a problem in itself.

DISCUSSION

DR. G. L. WALLACE: It is impossible for any of us to listen to these papers without being impressed with the importance of the subject matter. A few years ago it would have been difficult, in fact I think impossible, to get so many angles of the work. Dr. Myerson's paper impresses on us the way the subject of feeblemindedness is gradually being elucidated and set in a field by itself. His paper has shown us that it is a subject apart, in a way, from mental diseases. In Dr. Elkind's paper the unclassified group looms up as an extremely important social group. It carries us back a little over the road we have traveled recently of failing properly to emphasize the importance of the hereditary factor in mental deficiency and makes us wonder whether, in these last few years, we have not treated a little lightly that idea to the extent of minimizing the custodial care of the feebleminded. In the second paper we were impressed with the character of the diversity of that middle group of brains as symbolizing the middle group of the mentally defective, which becomes the real social problem. The lower group as a social problem does not exist, at least in a broad way, neither does the higher group. Dr. Healy's paper deals with the practical side of the work, and I was interested in the statement he made several times of not being able to place patients in the institutions for feebleminded whom he believed should be placed there. Right here I can but ask whether we have not been expanding too much recently the idea of community care to the exclusion of institutional care. When a patient enters an institution we do not want the professional interest to cease. We are using many of the known psychologic tests, but they give us little information from the practical side. What we want is to have some one tell us how to arrive at the goal of determining the dependable class of feebleminded in a quicker and more direct way. It often takes us years to make this determination. In our institution we find groups testing exactly the same mental age who are wholly dissimilar in their behavior reactions. For instance, we have the honest group with an intelligence quotient of 60 or 70; we also have the dishonest group with the same intelligence quotient. We have the temperamental group with an intelligence quotient of 60 or 70; we also have the rather stolid, reliable group with an intelligence quotient of 60 or 70. It is our business to ascertain what group is the reliable group; what group is the dependable group; what group is the one that will probably get along all right in the community and be an asset, and what group, of the same mental age level, is going to become a liability. We bring to our aid not only the known psychologic methods but also the practical methods of observation as to the way these patients conduct themselves, and from these methods we get accurate data and make concise and precise records. We go everywhere that the child goes. We get the reaction from the time the child gets up in the morning, in his classes, in his industrial activities, in his work about the school; and we observe how he sleeps, whether he has night terrors, whether he sleeps soundly, whether he sleeps with his mouth open, etc. Our institutions are

laboratories for studying the children, but we should like to know how to obtain, in a shorter time, the results that we do obtain by long and arduous study.

DR. O. J. RAEDER: I have heard what Dr. Myerson has said about syphilis among the feebleminded, and I do not agree with him. He made the statement that syphilis has nothing to do with feeblemindedness. I do not think he is right about this. I refer to a piece of work I did when I had charge of the syphilis clinic at the Psychopathic Hospital in which we examined thirty children of syphilitic parents. In this series there were two families in each of which there were three or more children. In the first family the first child had a positive Wassermann reaction in serum and a positive Wassermann reaction in the spinal fluid; the next child had a positive blood and a negative spinal fluid reaction; the third child, the youngest, had negative reactions straight through. Now Dr. Myerson will tell you that their parents may have been feebleminded, the father or mother, and as a result the children are feebleminded and just happened to have syphilis. In the next family one of the children was born before the father contracted syphilis, and that child did not have any trace of feeblemindedness or any signs of syphilis but was a bright, intelligent child of 16 years. The father came to America and contracted syphilis. His wife came over three or four years later and bore three more children. The first one of these three had a positive Wassermann reaction straight through; the second had a positive reaction in the serum and a negative reaction in the fluid; the third had a negative reaction in both the fluid and the blood serum. Here we have a family in which the first child, born before the father contracted syphilis, was perfectly normal; the next child was distinctly neurosyphilitic with all positive laboratory evidence, and the next one was feebleminded and showed a positive Wassermann reaction in the blood though the spinal fluid reaction was negative. This shows the attenuation of the virus by repeated pregnancies. Another question arises: How many of all the feebleminded children are in institutions for feebleminded? Not more than one or two of these patients we studied were in institutions for the feebleminded. Most of the families keep their children at home as long as they can, and only put them in institutions when forced. It is also well known that in children who give positive Wassermann reactions early in life there is a tendency for it to become negative as they grow older, so that older residents in institutions for the feebleminded will show fewer positive Wassermann reactions than younger children who are at home. In Germany, Weygandt and others who have studied this question find that if they take institutional patients under 10 years of age the number of positive Wassermann reactions is proportionately higher than if they take the Wassermann test on older feebleminded patients; 12 per cent and 14 per cent is common with these authors. Therefore, I believe that syphilis does play a rôle in the causation of feeblemindedness. I believe it is not a large rôle, though important, and must be considered in any general survey of feeblemindedness.

DR. WILLIAM HEALY: Is it possible that the population of institutions in this country, or in Massachusetts, represents a selection that vitiates statistical conclusions, or is the selective process that brings certain persons to institutions negligible in this sense? I have seen cases from time to time that make me wonder about various statements made on the basis of institutional experience, for instance, about the relationship of feeblemindedness of both parents to the mentality of their children. I have seen some children recently that come from a family, part of which is known to Dr. Thom. The woman has an intelligence quotient of 58; the man is feebleminded; all the children are

of distinctly good mentality according to mental tests; indeed, some of them rank as superior. Of course, I have been a little skeptical about the observations on the parents, although in this case the evidence is strong that both parents are defective.

My point in regard to special studies of the feebleminded is that these might afford much of value for educational psychology. I do not mean the ratings of defectives by age level tests, but rather the study of their capabilities in many directions and the differences in their powers of perception, learning, visualization, reasoning, etc. We are greatly lacking in a knowledge of the variations and combinations of these powers in normal persons, and the feebleminded with their simpler mental background and presumably smaller differences offer a chance for an easier program of study of the capacities and educabilities of the human mind. A rich field for educational psychologists to cultivate still remains largely untouched in our institutions for defectives.

Dr. H. B. Elkind: I should like to amplify what Dr. Myerson has said. We employed the statistical method in this study, not for the purpose of induction, but for that of description — to abstract from the material some of the hidden facts and relationships. A small number of the conclusions may have more or less universal application; but most of them are only suggestive. I believe most of them would be verified if we were able to make a similar analysis of like material from other training schools. We took the families in which we had adequate information and divided them into families in which there was only one sibling feebleminded (the patient), into families in which there were two (one other besides the patient) into families in which there were three (two others besides the patient), etc., and considered these families from the point of view of whether the parents were both feebleminded or whether one alone was feebleminded, or both not feebleminded. We found that 84 per cent of the families in which both parents were not feebleminded had only one feebleminded sibling; while only 11 per cent of the families with parents not feebleminded had five or more feebleminded siblings. On the other hand, when we studied the families in which both parents were feebleminded we found that only 5 per cent had only one feebleminded sibling in a family, but that 67 or 68 per cent were families in which practically all of the siblings were feebleminded. It seems to me that chance alone cannot explain these results; therefore, they may be considered somewhat suggestive. I feel, too, that similar studies obtained from other training schools, such as at Wrentham or Belchertown, would bring forth results approximately similar to those we have presented. Such additional studies are necessary before the conclusions presented in Dr. Myerson's paper may be considered as universal truths, before they can be accepted as possessing universal scientific validity. There is a selective process surrounding admission to training schools; there is no question about that, and our results should be viewed with this fact in mind. However, until one is able to analyze other data which are now becoming available, such as community studies into the extent of feeblemindedness, I think that conclusions derived from studies of material from training schools will probably give us the best available information concerning the inheritance of feeblemindedness. I also think that the information which Dr. Myerson has brought before us, that mental disease and epilepsy have nothing to do with the direct causation of feeblemindedness, has considerable scientific validity. Therefore, we may consider feeblemindedness as a unit character, in no way related to the so-called neuropathic or psychopathic constitution,

an assumption utilized by Davenport in his studies on the inheritance of mental disease and defect.

DR. MYERSON: I do not want to be misunderstood regarding the terms idiot, imbecile and moron. I do not quarrel with these terms as descriptive terms implying the degree of mental deficiency; what I emphasize is that they do not represent biologic classification. It is as if tuberculosis were not understood as to etiology and, therefore, tuberculous patients were described as large cavity types, medium cavity types and small cavity types. Since we have no known etiologic or pathologic factor in the most of feeblemindedness we are entitled to the use of the terms idiot, imbecile and moron, but in using them we must keep strictly in mind that they have no biologic, etiologic or pathologic meaning and represent a measurement of damage. Even as a measurement of damage they are, practically speaking, in one dimension only and give us no fundamental ideas as to feeblemindedness. In other words, they cannot be used in a paper which tries to be biologic in its point of view. The cases Dr. Raeder cited are not of any particular value because the number is too small to prove anything. If only 3.5 per cent of a population of over 3,000 is found to have signs of syphilis, there is not much room for the assertion that syphilis in itself causes feeblemindedness, since coincidence might easily account for that number. Our studies, which include the spinal fluid studies as well as an attempt at thorough clinical and familial investigation, show this at any rate, that syphilis, if it does cause feeblemindedness, causes it in only very small measure. The question has been raised as to the difference between our results and Dr. Davenport's. Dr. Davenport assumed the mendelian hypothesis, at least in his earlier writings, and then went ahead to prove the hypothesis. In order to do so, he included everybody in the family, even the most widely separated collaterals, and as pathologic characteristics bearing on feeblemindedness he included things which have no relationship to feeblemindedness, such as chorea, paralysis, criminality, nervousness and the like. Necessarily, if you stretch the limits of the family wide enough, and if you make the types of pathologic characteristics very inclusive, you can prove that almost anything is mendelian. You can prove that red hair has something to do with feeblemindedness. We do not know that the parents or relatives in our cases were feebleminded. We did not make psychologic examinations of them. We judged them by the community evaluation of them, by the family doctor's statement, by the statement of social workers, by their life history, by a dozen and one things which may be wrong. We did the best we could, and confirmed our reports in as many ways as possible. We think we found that a large number of people are feebleminded who have no heredity to account for it; we think we found that when one parent is feebleminded, there is an increasing amount of feeblemindedness in the descendants, and when both parents are feebleminded, there is a tremendous amount of feeblemindedness in the descendants. Dr. Elkind tends, on the whole, to interpret this as showing some degree of mendelianism in the hereditary cases. There I differ with him. No matter what type of heredity is involved one would expect more feeblemindedness when both parents are defective than when only one parent is. Assuming blastophoria, if the germplasm is injured on both sides, feeblemindedness should be present. The hypothesis which I am now trying to prove is that the involvement by some environmental force injured the germplasm. What that environmental force may be is utterly beyond my conjecture at present.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, April 6, 1926

ISADOR ABRAHAMSON, M.D., *President, Presiding*

THREE CASES OF INTRAVENTRICULAR HEMORRHAGE. DR. IRVING J. SANDS.

The first case was that of a man, aged 32, of excellent family and personal history. He was well until one month prior to his admission to the hospital. At that time he suddenly fainted while at work. He went home and returned to work on the following day, complaining for a week of headache and nausea. Two weeks later he fell unconscious, and was in a hospital for a week. He then complained of dizziness and headache. One week before presentation, he again became unconscious and was taken to the hospital, where he showed a bloody spinal fluid, rigidity of the neck, slight Kernig sign and diminished reflexes. After twelve hours he regained consciousness, and complained of headache spreading from the base to the vertex of the head. The symptoms gradually disappeared. He was recommended for discharge, the diagnosis being subarachnoid hemorrhage due to rupture of a small intracranial aneurysm. He suddenly lost consciousness, had peculiar adductor and abductor movements of his left extremity, and died. At necropsy, there was found a sacculated aneurysm of the left posterior communicating artery of the circle of Willis, which had ruptured in two places, the last time into the left inferior horn of the lateral ventricle, flooding the ventricular system. No other pathologic condition was found elsewhere in the body.

Case 2 was that of a middle-aged man who for about six months prior to his illness had complained of dizziness and headaches. He discontinued work two months ago. He suddenly collapsed while at dinner, frothed at the mouth, and became unconscious. He was taken to the hospital, where he showed definite cervical rigidity, fixed pupils, trismus of the jaw muscles and bloody spinal fluid. He died within one hour of admission to the hospital. He showed marked basilar cerebral arteriosclerosis, with hemorrhage into the pons, the fourth ventricle and the cisterna magna. He also showed chronic nephritis, general arteriosclerosis, and emphysema.

The last case was that of a school superintendent of about 60 years of age, who became unconscious while at work and was immediately brought to the hospital. He was observed by us for eighteen hours, and a diagnosis of intraventricular hemorrhage was made on the basis of the cervical rigidity, generalized spasticity, diminished deep reflexes, and the persistent bloody spinal fluid. He showed an intraventricular hemorrhage, but the source of the hemorrhage could not be located despite careful examination of the entire cerebral vascular system. He also showed cardiorenal vascular disease of the sclerotic type.

DISCUSSION

DR. E. D. FRIEDMAN: Have you any idea concerning the etiology in the first patient? There was neither hypertension nor evidence of infective endocarditis producing a secondary mycotic aneurysm of the brain. Did the patient show any evidence of status lymphaticus?

DR. SANDS: No; there was no evidence of status lymphaticus; he had no other pathologic condition in the body. About a third of the cases described do not show any reason for the occurrence of the disease. The other aneurysm that I presented a year ago did not have any. This case illustrated an inter-

esting point: the first blood count was 24,000 leukocytes. This high count is almost pathognomonic of cerebral hemorrhage in the acute stage. Four days later it fell to 14,000. In the case which presented the marked arteriosclerosis I could not find any cerebral aneurysm.

PITUITARY DYSOSTOSIS. DR. ARTUR SCHUELLER.

Lantern slides of roentgenograms were demonstrated of cases presenting defects in the skull of an unusual type called "pituitary dysostosis."

The first case was seen in 1914, in a girl, aged 5, who presented a left-sided exophthalmos. She began to drink 6 or 8 liters of water a day. This was followed by exophthalmos of the right eye, and then the defects in the skull became evident. They were sharply outlined. At the base, the floor of the sella and the upper part of the sphenoid body were destroyed. The top of the orbit showed in the plates a large defect with a resulting mechanical exophthalmos. The brain pressed on the eyeball and pushed it forward. The patient was again seen in 1919, at which time the defects had absolutely closed. Diabetes insipidus was present. The child had no symptoms of brain lesion; the intelligence was good.

The second case of this kind was published by Christian (Boston). His patient was also a child of 5 years with diabetes insipidus, exophthalmos and a large defect in the skull. I presume that this syndrome (diabetes insipidus, large defects in the skull, and exophthalmos) is due to a hypophysial disturbance. Christian thinks, too, that it is an unusual type of hypopituitarism.

One similar case published in Germany at necropsy showed atrophy of the pituitary gland; there were no cells; it was considered a case of pluriglandular insufficiency.

A similar case occurred in an Italian, aged 23; his symptoms began at 13 with exophthalmos and diabetes. The man did not grow from his thirteenth year, and showed multiple defects in the skull, so that we have again the combination of diabetes insipidus, exophthalmos and defects of the skull, together with dwarfism.

The next case was seen by me a few months ago. The child had diabetes insipidus, exophthalmos and large defects in the skull consequent to infectious disease. The lower jaw was gone. Later the defects disappeared. This also was a case of dwarfism.

The next pictures showed no defects in the skull, but a large area of osteoporosis. One patient had intense headache and was operated on by Dr. Cushing.

The remaining pictures were of skull defects presented for differential diagnosis. The first was that of a child, aged 12, with a defect of the vertex, with no symptoms of syphilis or tuberculosis. Following roentgen-ray treatment, the defect disappeared in a few weeks.

The second case was that of a boy, aged 15, showing defects in the parietal and temporal regions. He had an angioma of the skin; it was supposed that these defects of the skull were due to angiomas of the skull.

The third case showed a defect of the skull due to tuberculosis. The next was due to carcinomatous invasion of the bone of the skull, starting in the scalp. The next was a case which metastasized from a sarcoma of the testis; the next, a case of syphilitic osteoperiostitis; the next, one of multiple gummas of the skull; this woman was supposed to be an hysterical patient, but she showed multiple gummas, and after iodide treatment the defects were healed.

The following case illustrated a localized gumma. The next was one of myeloma of the skull with many small rounded defects; and still another showing metastases of a thyroid carcinoma to the skull.

"Pituitary dysostosis" seems to be largely an American disease; there are in the literature other cases described in America, by Hand, in 1903, and by Kay, in 1905. It was not supposed that these were cases of disturbance of pituitary gland secretion. Hand supposed his was a case of tuberculosis, and the other one was thought to be hydrocephalus. Grohs and Stifel (Toledo) in 1923 described a case; and in the last months, one was reported from Omaha. Apparently this pituitary disease is not as unusual as is supposed, and it seems to be more common in America than in Europe.

DISCUSSION

DR. LEOPOLD JACHES: I have seen only one such case, a few years ago. I believe it will be published by Dr. Denzer in whose patient it occurred. There were large defects all over the skull, and the child had diabetes insipidus.

DR. MOSES KESCHNER: Did the other bones show any other changes?

DR. SCHUELLER: The defects were mostly in the skull; but they were also found in the femur and in the pelvis.

DR. KESCHNER: Did the terminal phalanges show any evidence of the lesions observed in some of the other diseases of the pituitary gland?

DR. SCHUELLER: No.

DR. WALTER M. KRAUS: I have been very much interested in Dr. Schueller's paper, particularly in the relation of the bony changes in the skull which he has described to those which occur in the pituitary gland. The relation of the pituitary gland to the central beginnings of the visceral nervous system in the region of the tuber cinereum about the third ventricle has also been of great interest to me.

About ten years ago I presented a case of a Paltauf dwarf to this Society. In this case trophic disorder was an established fact in that the patient, although aged 37 years, was extremely small. The control of the water metabolism was conspicuously impaired, since this patient developed what I called a diabetes insipidus which ended in a complete anuria prior to death with preceding severe edema. Such transient edema was reported by Paltauf in his original description of this type of dwarf.

In addition to these findings the patient at necropsy showed almost complete destruction of the pituitary gland. The relation of the pituitary gland to the central beginnings of the visceral nervous system was not clear to me at that time. Since then the significance of this relation has been very much emphasized in many parts of the world, and Greving has recently shown that there exists a tract from the periventricular nuclei directly to the pituitary gland. Consequently, when considering the relation of the various signs and symptoms which are attributed either to the pituitary gland or to adjacent nervous centers, one must bear in mind that the origin of such signs and symptoms, including the skull changes which Dr. Schueller has so admirably demonstrated, may be only nervous and not pituitary in origin.

DR. B. KRAMER: Were there any changes indicative of the presence of late rickets? Were any blood studies made to determine whether there was a change in the condition of the blood-forming elements, and was any particular treatment effective in correcting this condition?

DR. SCHUELLER: In reply to Dr. Kraus, I suppose when we say "pituitary disease" we mean disease of the pituitary gland and the base of the third ventricle. It would be better to speak of the "pituitary system."

In reply to Dr. Kramer, the patients did not have symptoms of rickets. The bones were normal in shape, size and structure. Examination of the blood was made by Christian especially, and in the case published from Omaha. No disturbance in the blood was observed. No treatment was successful. We do not know why in one case the process progresses and in another the condition is cured. Diabetes insipidus can be influenced by treatment, by the injection of solution of pituitary. One of my patients had a good result from roentgen-ray treatment and iodine. The second patient showed improvement after measles, the defect beginning to close. It would be interesting to find the primary cause of the disease. I suppose there can be different primary, acute or chronic infectious processes invading the pituitary system. The importance is only in the localization of the process.

DR. ISADOR ABRAHAMSON: I would like to relate a case which presented many similar symptoms. The child was operated on for adenoids. The mother said that the child bled very much after the operation, a mass of adenoid tissue being removed. I saw the child about four weeks later with multiple malignant tumors over the body. What was operated on originally was probably a nasopharyngeal malignant tumor; the child developed bilateral exophthalmos; and had large masses throughout the skull, and one in the spinal column giving rise to a transverse myelitis; but the skull picture and the bulging eyes that Dr. Schueller described were present — probably a lymphosarcomatosis throughout the body.

Do children get over the exophthalmos or do they lose their eyesight? Is Dr. Schueller familiar with the further clinical histories of the patients?

DR. SCHUELLER: Some of the patients died, so that it is not possible to say anything about the course. The child in whom the defects were closed showed a persistent exophthalmos, and the other symptoms, diabetes insipidus and dwarfism, did not disappear. There are only eleven cases on record, and therefore one cannot say what the course of the disease will be, but the defects disappeared in many of the cases.

DR. ABRAHAMSON: There is a clinical syndrome called dysostose cleido-cranienne.

DR. SCHUELLER: We have seen these, because Scheuthauer of Vienna was the first to describe it. We have three specimens in Vienna. I did not show these cases because they are different. The defect is always in the median line corresponding to the region of the large fontanel and the shadow of the skull is characteristic. It is broad and flat. We saw two patients who had no other symptoms. They did not have diabetes insipidus.

FAMILIAL CALCIFYING CEREBRAL DEGENERATION. DR. WILDER PENFIELD and DR. H. RAWLE GEYELIN.

The case presented was that of a boy, aged 10 years, who since his fourth day had suffered from three different types of epileptic manifestations. The chronologic order of their appearance was as follows: (1) He had left facial spasm accompanied by bilateral pupillary contractions, occurring only when he ate, followed in two months by (2) generalized epileptiform convulsions which had diminished greatly in frequency during the past year; prior to that they were of daily occurrence. (3) At the age of 9, he began to have the

jacksonian type of seizure involving the right hand with the face turned to the left, followed by a brief period of mental excitation.

The family history revealed the mother to be in good health. There was temporary insanity at the birth of the last child. The father had had migraine headaches most of his adult life, but none since the occurrence one and a half years ago of three isolated major epileptiform seizures. He was of a lower grade mentality. Roentgenograms of the skull showed calcified areas in the brain. The oldest child, aged 15, showed essentially the same condition as the patient. The second child, a boy, died at the age of 4 with the same condition as that of the patient. Necropsy revealed multiple calcified tumors and an abnormally heavy brain. The third child, a boy, was the present patient. The roentgen ray showed calcified areas in the brain. The fourth child, a girl, aged 8, had convulsions similar to those of the patient which began at the age of 6 months. She also showed calcified areas in the brain similar to those of the father and older sister. All the children showed marked mental deterioration.

The results of the general physical examination of the patient were essentially negative. The blood and spinal fluid Wassermann tests were negative. The general neurologic examination showed no clinical localizing signs. The values for the blood and spinal fluid calcium were within the normal range. The calcium excretion in the urine and feces over a fifteen day period was within normal limits. The basal metabolism was normal. The routine blood and urine examinations gave negative results.

Stereoscopic films of the skull showed a dense, irregular shadow in the right posterior parietal region which seemed to be parallel with the inner table, but which projected into the cranial cavity. It appeared as though the calcium deposits were within the brain substance rather than in the meninges. The mass was approximately the size of a pigeon's egg.

When the boy came into the hospital, he had a palsy of the left sixth cranial nerve; the left pupil was larger than the right. The optic disks showed a moderate amount of primary atrophy; otherwise the physical examination revealed nothing of importance. The mental condition was unsatisfactory. While in the hospital, his mental condition became steadily worse, so that when asked to put out his tongue, he only opened his mouth or did nothing. Roentgenograms of the skull showed in the occipital lobe on the right side a calcified area which was more or less discrete. There was evidence in the plates that it was either hollow or that there were two furrows. There was calcification also in the region of the pineal gland. It was thought that it might be a calcified pineal gland pushed across to the opposite side by the expansion of the lesion in the occipital lobe.

After obtaining the history that the older brother had died with multiple calcified tumors, roentgenograms were taken of the rest of the family. Roentgenograms of the mother's skull showed nothing abnormal. The father's skull presented three calcified areas in the brain and some smaller ones near the surface. These shadows were either umbilicated or hollow. Roentgenograms of the sister showed three calcified areas in the cerebral hemispheres which also evidently were hollow. For these roentgenograms we are indebted to Dr. Weeks, Superintendent of the Village for Epileptics at Skillman, N. J. The sister, Elise, aged 8, showed many scattered calcified areas throughout the brain, with definite furrows or hollows in them. These were all placed at a distance from the base; none were in the brain stem. They were all at the maximal distance from the circle of Willis; none appeared in the cerebellum

and in general possessed the characteristics shown by the roentgenograms of the other three cases.

In regard to the operative case: Drs. Penfield and Geyelin were unwilling to operate at first, realizing that the condition was a congenital one. However, they had the necropsy diagnosis from the hospital in Elizabeth, N. J., that the oldest brother had had multiple calcified tumors. The patient under consideration was the only one who showed calcifications localized to one point in the brain, and the calcified pineal gland was apparently pushed to the opposite side. His general condition was extremely unsatisfactory. Therefore the patient might possibly have a calcified tumor which was cystic. The prognosis was not hopeful. At operation a bone flap was turned down over the right occipital lobe. The dura was normal in appearance. On turning back the dura there was an area corresponding to the large discrete nodule seen by roentgen ray which was quite avascular. There were almost no vessels on the surface of the brain here. The tissue was hard on palpation, and one could feel the sulci as resistant and rubbery. Anteriorly the brain appeared normal. Posteriorly it was normal except for two areas where there was a little induration. As there seemed to be a line of cleavage between the brain and the indurated areas the whole occipital lobe with the tumor mass in it was resected.

Pathologically the case was of extreme interest. In the gross there was an egg-shaped mass measuring 8 by 4 by 3 cm. The cortex toward the midline from it was a coral pink. It was definitely rubbery and resistant. In cutting through the mass the knife met with a resistance which felt like soft sandstone. In the gross one could see a calcified zone which encircled a fissure of the brain, placed at the junction of the white and gray matter and running up into the gray matter a little distance. In the cortex posteriorly there was little except small hemorrhages which undoubtedly occurred at the operative removal of the specimen. It may be significant that the location of these small hemorrhages corresponded in a general way to the situation of the calcium zone more anteriorly.

In the brain tissue at a distance from the calcified areas there were corpora amylacea which occur not infrequently in older patients in the substance of the brain. They were located mostly in the vicinity of the vessels.

The zone of calcification was made up of rather loosely arranged fibrous neuroglia cells among which were small granules of calcium. The vessels showed the most striking changes. The capillaries were in many instances converted into tubes of calcium. The small arteries and veins were also calcified. Most frequently the calcium was laid down in the media; it was sometimes in the adventitia also, and then there was great thickening of the media, so that the vessels might be closed entirely. Some of the capillaries were closed. At a little distance from the calcified area in the substance of the brain there was a marked glial reaction. It was patchy and frequently arranged around vessels and made one think that the reaction in the brain itself was secondary to closing of the vessel. Elsewhere giant neuroglia cells were seen, some of which were frankly undergoing degeneration and dendrophagocytosis. From a histologic point of view the evidence was in favor, not of a parenchymatous change of the brain, but of a primary change in the vessels; the evidence of the neuroglia changes particularly pointed in that direction.

After operation the patient showed a complete left hemianopia; he had a left sixth palsy which was not always present and a paresis of the right facial nerve which showed in voluntary but not in emotional activity. Other-

wise, physical examination revealed nothing abnormal. The month after he went home, his mother said, was the best month of his life; he improved mentally rapidly and had no more convulsions. It was a much more favorable result immediately after operation than often is procured with the removal of a brain tumor, but Dr. Penfield did not urge this treatment for the condition. During the last month or so, the patient had again a return of the convulsions, although less intense. He still was very much better mentally than he had been.

Physical examination of the little girl revealed nothing abnormal; she had a left sixth palsy which appeared for one or two days and then disappeared.

From the histologic point of view, there was evidence of a primary change in the vessels of the brain which was situated at the juncture of the superficial pial vascularization with the deeper vascularizations. It occurred in the deeper layers of the gray matter and extended down to the white matter. It was thus located for the most part near the end-arteries, and in one sense might be called an obliterative endarteritis. Roentgenograms of the other cases have shown that the calcifications are in the cortex of the brain, not at the base, but at a considerable distance from the circle of Willis and often, at least, situated about fissures in a manner similar to that described above.

Without going into the literature fully at present, there are two chief types of calcification. Rokitansky has shown that calcification of the brain may occur; the nerve cells may be calcified and the arteries may be calcified, particularly in the white matter. Virchow pointed out a condition which he called metastatic calcification which occurred as the result of acute destruction of bone, anywhere in the body, as from tuberculous caries or osteomalacia, and it has been shown experimentally that the injection of calcium will produce these calcium deposits. It is deposited in certain points of election, that is, in the stomach, the lungs and the kidneys particularly. Wells pointed out that these are the organs which are probably most alkaline, as they are secreting or giving up acid and it is possible that a decrease in carbon dioxide in the blood is a cause for the depositing of calcium in these areas.

In this group of cases there is justification for concluding that the deposit is not due to an increased amount of calcium in the blood, as the roentgen ray showed no calcification in the thorax or abdomen where the sites of election were located. Everywhere in the body degeneration or necrosis will, in time, produce calcification. It seems that we are dealing here with a degeneration secondary to the obliteration of localized end-arteries, this degeneration becoming calcified. The condition certainly is familial, for it occurs in the father and four children. On a mendelian basis, the process must be in the mother's family as well. It seems quite likely that it could be, because of the probable amount of inbreeding in a small town of the sort she came from. The husband came from a small town not far distant. The roentgenograms seem to present a new type of appearance characterized by the laying down of calcium about the fissures of the surface of the cerebrum, and it should be possible to diagnose this condition simply from the roentgen-ray findings.

Although the condition of the boy who died was diagnosed as calcified tumors, there was some doubt about it at the time, and every effort was made to secure the specimen. The hospital had moved; the pathologist had died; the technician who cut the sections had died, and the specimens were lost. Therefore there was only the diagnosis written on the necropsy slip.

From the point of view of tuberous sclerosis there was no evidence of adenoma sebaceum, and there were no areas which suggested glioma in any way. There was a marked gliosis, but in no sense glial cells which characterized new growth.

DISCUSSION

DR. B. KRAMER: About a year ago I saw a child who was brought to the dispensary for examination because of convulsions and fever, and all we could find were signs of cardiac hypertrophy. The child was admitted to the hospital and died in convulsions. Necropsy showed a tremendous heart and extensive calcification of all the blood vessels, including the coronaries. I would like to ask whether there was any evidence of renal insufficiency in these cases, and whether there were any pathologic changes in the kidneys in the fatal case.

We have been interested for some time in the problem of calcification, which may be divided into two parts. The first is, why does calcification occur; and second, why does it select certain places? We have been able from our studies on calcification in rickets and its failure to occur in ununited fractures to throw some light on why it occurs normally. We have shown that calcification will not occur unless the bone-forming elements are in sufficient concentration in the blood. When they exist at a minimal level, calcification will not occur, and it will occur just as soon as the concentration reaches the proper level. As to why it should occur in certain places, and not in others, we have some evidence, but it does not solve the problem. One factor is the blood supply. It is striking, as Dr. Penfield has shown, that calcification occurs in the zone just between pial blood supply and the vascularization from below. Calcification occurs in the provisional zone of calcification in cartilage in the zone between the blood vessel coming from the perichondrium and going down to the juncture of the epiphysis and diaphysis, and those from the shaft that go up through the marrow spaces. It is in the zone between these terminal vessels that the calcification occurs. If you look at the calcification process as it occurs in the trabeculae of the bone, you will find that the blood vessels come in contact with the osteoid which lines the trabeculae, but the calcification occurs in the center of the trabeculae and works its way outward.

DR. ROSS GOLDEN: The diagnosis of tumors of the brain by roentgen ray depends usually on the detection of, often, relatively slight changes in the bony structure. Calcification of brain tumors unfortunately occurs only rarely. When these films came up in the course of routine work, I assumed that we were dealing with one of these unusual calcified tumors. Dr. Penfield's operative observations came as an interesting surprise, especially in connection with the subsequently obtained histories and skull films of the other members of the family.

The most important thing from the standpoint of roentgen-ray diagnosis is to decide whether it is possible to distinguish these conditions from other calcifications which occur in the brain. A critical examinaton of the roentgenograms discloses a number of facts, some of which have already been mentioned by Dr. Penfield, but which are worth emphasizing: (1) the distribution of the shadows throughout the hemispheres; they occurred bilaterally in every case; (2) the situation of the shadows a relatively short distance beneath the surface of the brain; (3) the multiplicity of the shadows; (4) their character. Three types of shadows are present in these films: The first resembles the so-called "mulberry" shadow of calcified tuberculous nodes. If some of these were seen on a film of the abdomen or the neck, they would be interpreted as typical calcified tuberculous nodes. The second type is crescent or U-shaped. The third is ringlike or ovoid.

Bearing in mind the situation of the calcium deposits in the convolutions, as demonstrated by Dr. Penfield, it is obvious that the shadow will be ringlike,

ovoid or crescent shaped, depending on the relation of the mass of calcium to the direction of the beam of rays. In every instance the density is irregular.

I have never seen just such shadows as these in any other condition, and, in view of their correlation with the histologic findings, I believe that they can be considered as characteristic of this calcification of the brain. It is a most unusual condition and is certainly in marked contrast to the usual unfruitful examination of the skulls of epileptic patients by the roentgen ray.

DR. SCHUELLER: I am very much interested in these cases. I have seen a good many calcifications of the brain, but never anything like this. Two things are especially interesting: The first is the morphologic appearance of the calcification, and the second is the familial history. The type of calcification is what we see in a scar. There are two types — one in a tumor and the second in a scar. The most important point is that it is following the vessels of the brain. I think that the expression "tumor" should not be used. It is an old scar in the brain showing gliosis, thickening of the vessels, and calcification mostly around the vessels. The question arises: What kind of disease is it? It consists of familial scars in the brain showing calcification. I am reminded of periarteritis nodosa. This disease is unusual, and I would think it could be a case of familial periarteritis nodosa.

DR. GEYELIN: In answer to Dr. Kramer's question: Studies of renal function were made, and it was entirely normal. Rather complete calcium and phosphorus studies were made, not yet finished, but I am able to say that the calcium excretion in the stools and urine, over a fifteen day period, on a known calcium intake, showed no variation from the normal. The blood calcium and spinal fluid calcium before and after operation were within normal limits. The basal metabolism, tested on two occasions, was in the normal zone; it was plus six and plus four. There was nothing abnormal in the blood or elsewhere.

I think Professor Schueller's conception of the similarity to periarteritis nodosa is an extremely interesting suggestion.

As to the mentality of the father, he is able to earn a livelihood; I think he is slightly subnormal.

The points brought out by Dr. Penfield, both as to the microscopic character of these lesions and the roentgen-ray appearance, rather suggest that this is a unique condition. Whether it can be associated with any of the conditions spoken of, I do not know.

PHILADELPHIA NEUROLOGICAL SOCIETY

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WILLIAM G. SPILLER, M.D., *President, in the Chair*

THE EFFECT OF CORDOTOMY ON SPASTICITY, AND REACTIONS OF DEFENSE. DR. TEMPLE FAY.

Cordotomy, in its strictest sense, means a section of any portion of the cord, but in this country the term implies section of the anterolateral columns of the cord for relief of pain, unless otherwise qualified. In 1911, cordotomy was performed for the first time on a patient who was suffering from persistent pain due to sarcoma of the lower vertebrae. After operation, the patient's suffering was greatly relieved and pain and temperature sensations in the lower extremities were practically lost.

Dr. Frazier through a careful development of surgical technic placed the operation on a sound basis, so that many cases are attended with only a loss of pain and temperature sense, as intended, without a disturbance of the other functions and tracts of the spinal cord. Schuller, in 1910, suggested anterolateral section of the cord but this was not carried out until some years later. In 1912, Cadwalader and Sweet reported their observations of two cases of cord section on dogs. Hunter has thrown a great deal of light on the subject of muscle tonus by saying that it consists of two elements, "plastic tonus" and "contractile tonus"; the former has the function of maintaining posture, once the posture is assumed, and the other produces changes of position and posture. The theory seems to show the existence of a dual nerve supply to muscle fibers.

Sherrington claims "that tonus is a beautifully graded series of proprioceptive reflexes, continuously and unconsciously playing its part in our every motor act; by its remarkable specificity it moulds our individual muscles; by its universality it controls our postures."

REPORT OF CASE

A girl, with a diagnosis of cord tumor, suffering from weakness and paresthesias in the lower extremities progressing into spasticity of the left leg, with disturbances of pain and temperature, presented herself for treatment. A laminectomy was performed and an intramedullary tumor found at the seventh dorsal segment. After operation, besides having pain about the chest and abdomen with girdle sensation, the patient noted that she could not move her legs voluntarily though they were not as spastic as before operation. Three weeks after the operation the legs would involuntarily jerk upward. These contractures were frequent, and spontaneous clonus occurred on handling of the parts. In the two months following the first operation, reactions of defense were marked and present in both extremities by flexion of the big toe. They disturbed the patient's sleep because of their frequency and were associated with such pain that cordotomy was decided on above the level of the lesion. After a neurologic examination was made with the opinion that there was a cord tumor as high as the level of the fourth and fifth dorsal segments, probably intramedullary, a second operation, laminectomy and cordotomy, was performed. A gelatinous tissue in the cord thought to be a diffusely infiltrating glioma, partly degenerated, was found. Up to this time, the patient's knees, which had remained in spastic right angle contracture (a few seconds after the section of both anterolateral tracts), now gradually straightened themselves to an angle of about 150 to 160 degrees.

Two weeks following reexploration and cordotomy a decided change in the subjective and objective symptoms was noted. Impairment for heat and cold was more marked than before operation, pain sense was lost to a greater degree than temperature, and the reactions of defense could only be elicited with effort. Spontaneous drawing up of the lower extremities had disappeared, and pain was almost entirely relieved. One week later, it was found that the sensory observations were the same, but there was a return of spasticity and the reflexes of defense were quite marked. Involuntary contractures were present, though not to the same degree as before.

Five weeks later the reflexes of defense had become more marked. Spontaneous drawing up of the legs had returned to the same degree as before operation, though not associated with pain. Spontaneous clonus with spasticity was as marked as before cordotomy.

As an interpretation of the foregoing in the light of present experiments, Drs. Cadwalader and Sweet noticed not only a loss of pain and temperature

sense, but in addition an awkwardness of the hind legs in their animals, which also frequently misplaced the feet, fell or assumed peculiar positions. Unfortunately, as their observations were for pain and temperature determinations, no note on the reflexes was recorded, excepting that on the day following the operation, "dog cannot stand or walk on account of inability to use hind legs. Tendon reflexes are equal and active. There is no spasticity nor do the hind legs feel exactly flaccid." Spiegel published some studies of cordotomy on dogs and cats and noted that hypotonia was present for from four to five weeks.

In summarizing these experiments it will be found that, from the surgical point of view, section of the posterior columns can be undertaken with the least amount of trauma and section of this area is usually followed by hypotonia for only four or five days, with a return of tonus to the lower extremities. Hypotonia lasted for two weeks when the more difficult section of the anterolateral columns was undertaken, requiring manipulation of the cord and deep cutting of the fiber tracts. In the combined section requiring more profound disturbance of the cord, hypotonia lasted from four to five weeks. In each instance there was a return of muscle tonus after a varying interval. We must agree that recovery of automatic function occurs in the cord after we consider the results from the standpoint of interruption of the tonectic pathways, and the spinocerebellar and vestibulospinal, as well as certain different proprioceptive pathways.

Eight cases are reported by Frazier and Spiller. In one case there was a distinct hypotonia following section of the anterolateral columns, one month after operation. The patient died and a necropsy was performed. Degeneration of the peripheral portion of the anterolateral columns and of the anterior portion of the direct cerebellar tracts was found. In another case reported, some damage occurred to the crossed pyramidal tracts and on comparison of the reflexes before and after operation, some hypotonia existed.

In the case here presented, a tumor of the thoracic cord centrally situated produced almost complete physiologic interruption of the ascending and descending pathways. Tonectic regulating pathways were also greatly interfered with, as shown by the spasticity, increase in reflexes and reactions of defense. If any pathways in the anterolateral region of the cord escaped the tumor in their efferent and afferent course from the higher centers, they were probably completely destroyed by the liberal cordotomy performed above the level of the tumor. All function of the left side of the cord was lost after cordotomy. Hypotonia was manifest almost immediately in the relaxation of the lower extremities on the operating table, and the hypotonia was more marked on the right side than on the left following cordotomy. The former is difficult to explain on the basis of tonectic pathway destruction alone, because of the doubtful existence of functioning pathways either ascending or descending through the tumor mass, just below the level of the cordotomy. The return of spasticity and reflexes of defense two months after operation to their preoperative state, implies the ability of the cord to assume an automatic tonic regulating function; while the relaxation, if caused by spinal shock, would suggest the presence of functioning pathways capable of transmitting stimuli through the tumor mass to the cord below.

Before cordotomy the symptoms showed less involvement of the right half of the cord; following cordotomy there was evidence of function remaining on the right side. The final observations show a return of tonus and reactions of defense to their former state, and the sudden loss of tonus at operation, followed

by its gradual return, simulates the observation noted in traumatic lesions of the cord, in which, after two or three weeks when all reflex manifestations are lost, there is a gradual return of reflex activity.

In conclusion, whatever tonectic pathways exist in the anterolateral columns of man, section of these pathways in the dorsal region produces only temporary loss of tone in the musculature of the lower extremities, and the effect of cordotomy closely simulates spinal shock produced by trauma.

DISCUSSION

DR. W. B. CADWALADER: In 1911, Dr. Spiller suggested for the first time that cutting of the anterolateral portions of the spinal cord would serve as a practical method of relieving intractable pain, and he asked Dr. Sweet and me to try this on dogs to determine the effect on pain conduction. Accordingly we operated on a series of seven dogs. Two of them seemed to be technically correct.

Dr. Fay has just referred to our report, which was made before the College of Physicians and published in 1912 (*J. A. M. A.* **58**:1490-1493 [May 18] 1912).

The lantern slides show one of these dogs. Cutting of the anterolateral columns did diminish pain sensation. In addition there was a condition we termed pseudoparalytic state, with incoordination, involving the hind limbs. The dogs assumed curious attitudes, which we attributed to the inability to recognize the posture of the limbs; and we thought it might be due to the cutting of the afferent and efferent connections between the spinal cord and cerebellum, which are believed to traverse the anterolateral region of the cord. The hind limbs were probably hypotonic, yet in our report this particular phase of the subject was not discussed. But the important fact is that these apparent motor phenomena were not permanent; they disappeared entirely in less than two months. Therefore, we concluded that the condition was a manifestation of spinal shock and was not dependent on the cutting of any particular set of fibers whose special function it might be to control muscle tone or muscular coordination. Microscopic sections of the spinal cord prepared subsequently and studied by the Marchi and Weigert methods showed that only the anterolateral regions were destroyed; the degeneration could be traced upward, and the pyramidal tracts were intact.

Dr. Fay stated that in his case of paraplegia caused by spinal tumor, the hypertonicity, and especially the flexion reflex of the lower limbs, has been conspicuous and that immediately after cordotomy, which was successfully performed for the relief of pain, the flexion reflex subsided on the side on which operation was performed, and the limb became temporarily hypotonic; after some weeks had elapsed, hypertonicity and the flexion reflex reappeared and remained just as conspicuous as it had been before the cordotomy had been performed. He has attributed this to spinal shock, a view with which I entirely agree. In discussing these observations Dr. Fay has quoted the experiments of Spiegel (reported at the German Neurological Association in September, 1925) recently published in Germany, in which that author asserts that after cordotomy in cats and dogs hypotonia occurs in the muscles of the limb of the same side. Foerster, in discussing Spiegel's work, apparently accepts this conclusion, for he suggests that cordotomy would be useful as a means of diminishing the excessive hypertonicity of spinal cord disease or of extrapyramidal tract disease. These writers do not seem to know of our experiments for they do not mention them. Their results seem to resemble those which we had observed, but our interpretations are different. The condition Spiegel

refers to in his animals as hypotonia was not permanent. He says it lasted three or four weeks only. Judging from his descriptions, I am convinced that he observed a phase of spinal shock, and that the apparent hypotonia was not the specific effect of cutting certain spinal tracts whose function it is to control muscle tone. It is well known that there are variations in the manifestations of spinal shock, and that in spinal lesions which grow very slowly and produce spastic paraplegia gradually, spinal shock does not appear; yet all the spinal reflexes and hypertonicity may become highly developed, just as in Dr. Fay's case. On the other hand, spinal shock becomes conspicuous when the cord is transected suddenly, for example, by gun-shot wound. Many such cases were observed during the war. Shock has been defined as a transient depression of reflex activity, and it is asserted that the principal factor involved is the loss of impulse transversing the descending tracts of the cord. I believe, however, that the intensity of spinal shock depends chiefly on two factors; the amount of tissue destroyed and, most especially, the suddenness with which the destruction occurs. Any surgical procedure produces effects suddenly. In Dr. Fay's case the symptoms indicated that much of the cross section of the cord had been destroyed slowly by the lesion that was present. And then the additional and sudden destruction of the remaining cord tissue effected by cordotomy had produced a complete anatomic section of the spinal cord. That is to say, the equivalent of a spinal animal. The first effect was the diminishing of reflex activity or spinal shock, a temporary condition; for after a period of time reflex activity again became intense, just as it had been before the cordotomy had been done.

If it is true that destruction of the anterolateral columns of the cord, or cordotomy, has the specific effect of permanently diminishing the muscle tone in cases already hypertonic, then the reverse should also be true. That is to say: if the anterolateral regions of the cord are destroyed first and the pyramidal tracts are at a later period implicated, the development of the familiar spasticity and exaggeration of reflexes should then be prevented.

The following case with necropsy which was reported by Dr. Spiller in 1904 (*Univ. Penn. M. Bull.* July-Aug., 1905) clearly proves that spasticity and hypertonicity of the lower limbs may develop from destruction of the pyramidal tracts in the spinal cord, in spite of the fact that the anterolateral regions of the cord had been previously destroyed.

The patient had the symptoms of destruction of each anterolateral column of the cord when he was admitted to the hospital Aug. 31, 1904 — that is to say, loss of pain, heat and cold sensations in both lower limbs. The tendon reflexes and muscle tone were normal. There was neither flaccidity nor spasticity, and the man was not paralyzed. He was an ambulatory patient in the wards of the hospital. On September 16, just sixteen days after these observations had been made, he fell down stairs and was so severely injured that he was immediately and completely paralyzed in both lower limbs. Soon all the tendon reflexes became greatly exaggerated and there was ankle clonus and spasticity, and a bilateral Babinski sign. All these symptoms persisted until his death. The necropsy showed that the original lesion had been two separate tubercles, one in the right and one in the left anterolateral region of the spinal cord, corresponding exactly to the situation of Gower's tract. Degeneration of each pyramidal tract had been acquired subsequently by the injury and was, therefore, the younger lesion. The case is unique and important because it had proved the situation of the fibers for pain and temperature sensations in the anterolateral portion of the spinal cord; and in my opinion it now serves as proof

that destruction of the anterolateral columns of the cord in man does not prevent the development of spasticity and exaggeration of reflexes in the lower limbs from destruction of the pyramidal tracts.

COMBINED GLOSSOPHARYNGEAL AND CERVICAL POSTERIOR ROOT SECTION, FOR PAIN FROM CARCINOMA OF THE THROAT. DR. TEMPLE FAY.

A white, married woman, aged 41, had been under treatment for nine months for primary carcinoma of the tongue and soft palate, on the left, and had extreme pain, deep in the ear, behind the ear and in the throat. Two months before, she had a sphenopalatine injection, as well as extensive radiation, but as the growth extended there was constant pain, associated with great difficulty in swallowing.

Because of the extreme pain deep in the ear, and its exacerbation in swallowing, as well as the pain in the cervical distribution, a combined cervical rhizotomy and intracranial section of the ninth nerve was undertaken.

There were three points of great interest in the case. First, the fact that by means of rectal anesthesia, the operative procedure was made possible to a satisfactory degree, as there was complete anesthesia throughout the entire procedure. Second, the ninth and upper cervical posterior roots were included in the preparation of the surgical field, this being accomplished by a midline incision, exposing the upper three cervical laminae, after removal of the atlas, axis and part of the third cervical lamina; the upper cord was disclosed and then an incision was made at right angles to the midline incision, carrying it well to the left, almost to the mastoid and sufficiently below the superior occipital ridge to avoid injury to the occipital artery. The skin and muscles were sectioned in one block. The occipital bone was removed over the left cerebellar hemisphere, along the margin of the foramen magnum to the point of entry of the vertebral artery. The third point of interest was the dural incision which was devised for this procedure and consisted of a fish hook shaped opening. By means of this particular incision it was possible to see the structures beneath the transparent arachnoid, and to trace, by means of the spinal portion of the eleventh nerve, its course as it proceeded upward to emerge from the jugular foramen, where at its point of emergence it was immediately joined by the tenth nerve, which was composed of several fan shaped filaments. A small structure the size of a match stick was recognized as the glossopharyngeal, also making its emergence at this point. The ninth nerve was isolated, the arachnoid punctured, the nerve secured on a hook and evulsed. The upper two cervical roots were isolated, crushed and destroyed. The operation was almost entirely extra-arachnoid, and no bleeding from the outer wound reached the subarachnoid space.

The patient made an uneventful postoperative recovery, the wound healing by first intention. The relief of pain was marked in this case.

Anesthesia was established over the left posterior aspect of the scalp, below the ear, and a disturbance for pain sense even under the angle of the jaw anteriorly as well as onto the cheek. The left side of the soft palate and pharynx was also anesthetic. The patient for the last two weeks noted twinges the third division of the fifth which requires alcoholic injection to insure complete anesthesia. The patient has resumed eating and has gained 12 pounds (5.4 Kg.).

It is interesting to note, now that it has been possible to remove the sensory supply of the fifth, ninth and cervical nerves, that there still remains an area

which retains sensation. This must be, therefore, from either the seventh or the tenth. This case is unique in the combination of cervical and glossopharyngeal destruction and offers a means of application of this type of surgery to conditions involving the distribution of the ninth and cervical nerves. The exposure discloses the cerebellopontile angle and need not open the arachnoid, thereby excluding blood from the subarachnoid space.

This case is one of seven in a series of cervical rhizotomy, but the only one in which the ninth nerve was included with destruction of the upper cervical posterior roots.

A Case of Thalamic Syndrome with Homonymous Hemianopia and Aphasia. Dr. C. A. Patten.

This case is presented because the localization of the lesion is somewhat unusual and difficult. The patient shows a dysesthesia of the right side of the body, a right hemiparesis, right homonymous hemianopia and irritative phenomena. The patient is an Italian, aged 42, a laborer, married and has five children. His previous history and family history are unimportant.

In November, 1923, after working all night and while attempting to shave, he suddenly became weak in the right arm and leg, and was unable to talk. He could not see clearly and had a peculiar taste in his mouth. He believes he lost all sensation on the right half of his body. He was not unconscious but remained in bed for twenty-four hours. The paralysis of the right side quickly cleared up as he was able to shave on the following morning without difficulty. There remained, however, a marked motor aphasia and paresthesias of the entire right half of the body. The paresthetic phenomena consisted of peculiar burning, cold and prickling sensations which have persisted to the present time. In addition, there have been, since the onset, daily attacks in which the paresthesias become more intense, the speech more difficult. These attacks begin in the neighborhood of the right ankle, the peculiar sensations ascending in wavelike manner over the leg, thigh, trunk, face, tongue and then the right hand and arm. When the face is reached, it twists to the right and the tongue is drawn to the right. Following this, the right hand and arm twitch and if the patient is holding anything in his right hand at the time, it is dropped. On two occasions he has lost consciousness, but for the most part he knows what is going on around him during the attack.

He came under observation eight months after the onset of the disease, and at that time showed, from a physical standpoint, overweight, flushed face and blood pressure 120 systolic and 60 diastolic but no other physical symptoms or signs. Neurologically, there was a right homonymous hemianopia, weakness of the lower right face, lowered temperature of the right arm and leg, right biceps reflex more active than the left, abdominal reflexes absent, knee and ankle reflexes active and equal and no clonus or Babinski reflex. Pain, touch and temperature sensations on the right side of the body were preserved but he stated that there was always a difference between the two sides, in that any stimulus applied to the right side produced a disagreeable sensation. The muscle and joint sensations were normal. Speech was distinctly aphasic, words of one syllable causing great difficulty. He distinctly stated that he knew what he wanted to say but was unable to "get it out" of his mouth. He recognized objects and their uses. He was able to write his name and to pick out numbers on a printed page. He could copy without a mistake from a written page. He could write numbers on dictation. He obeyed commands readily provided they were not too complex. Some difficulty was experienced in making

tests for sensory aphasia because of the patient's illiteracy and inability to use the English language. On the other hand, it was noted that there was, in addition, some failure of comprehension of the Italian language. Since coming under observation, there has been no definite progress in the symptoms.

Laboratory investigation showed a negative blood Wassermann reaction and a negative spinal fluid. The Bárány test gave no conclusive evidence but responses which might indicate a lesion of the cerebrum. A roentgenogram of the skull was negative. An encephalogram, made recently, showed a shrinking of the left cerebrum with a dilatation of the ventricle on the same side and a displacement of the ventricles somewhat to the left.

The suddenness of the onset of the symptoms and the fact that there has been no increase in symptomatology in nearly three years point to a vascular lesion. The area involved must be either cortical or subcortical with implication of the prethalamic fibers, the optic radiation and the motor pathway in the left cerebrum. The occurrence of jacksonian attacks with sensory and motor manifestations is probably in favor of a lesion which, if not located entirely in the cortex, begins subcortically and reaches the surface. A thrombosis of some branch of the middle cerebral artery is predicated.

DISCUSSION

Dr. Alfred Gordon: If it is a thalamic lesion, how does Dr. Patten reconcile the fact that there is no involvement of the deep sensation? I believe that the patient presenting a right-sided paralysis and right homonymous hemianopia has a lesion in the optic radiations of the opposite side. The fact that he has convulsive seizures does not remove this possibility. In fact, any lesion in any part of the brain is likely to bring on a convulsive seizure. But the fact that deep sensibility is normal makes me hesitate to accept the thalamic view.

Dr. Charles K. Mills: It seems to me that the history and symptoms of the case point to a suprathalamic lesion in the corona radiata, this lesion extending back to the occipital lobe. The hemianopia would be caused by a lesion of the optic radiations. I do not think that there is any proof of involvement of the thalamus itself.

Cortical Lesion Producing Segment Paresis with Pain. Dr. Joseph McIver.

This case is presented to the society because the patient has a cortical lesion on the left side and a paresis of the right thigh, with pain.

The patient, a white man, aged 33, says that he was struck on the left side of his head with a blackjack one year ago. He was intoxicated at the time he received the blow and was unconscious for some time following the injury. He was confined in a hospital for three months, and states that he remembers nothing of the first two months of his stay there.

After two months in the hospital, he was allowed out of bed. At this time he found that his right thigh was weak, stiff and painful. He went about on crutches for eight months, at the end of which time he was able to walk without them. It was at this stage that I first saw him.

Examination.—He was distinctly lame in the right lower limb when he attempted to walk. The thigh muscles were spastic, but the leg muscles were not spastic, and he complained of a sharp, burning pain, extending from the hip joint to the knee. The pain was not continuous but came and went; walking did not influence it.

All attempts, including repeated roentgen-ray examinations, have failed to locate any trouble about the sacro-iliac, hip or knee joints. There was no

area of tenderness in the painful area. The adductor group of muscles were particularly spastic, and sudden forced abduction of the thigh would produce pain. Voluntary abduction of the thigh was distinctly limited but did not produce pain. The achilles reflexes and the left patellar reflex were moderately increased, but the right patellar reflex was markedly increased. There was no Babinski reflex. Sensation for pain, touch, temperature, vibration, position and passive movement was normal throughout. Wassermann reactions of the blood and spinal fluid were negative. An eye examination revealed a pallor of the optic disks and a contraction of the visual field. This might be alcoholic in origin. The patient did not complain of headache and there was no vomiting.

There was a distinct depression in the left parietal region of the skull about one and one-half inches long and about one-half inch wide. The roentgen-ray examination did not show any depression of the inner table of the skull. The lesion was located at a point which would represent the cortical thigh center for motion and sensation. For this reason it seemed reasonable to conclude that the pain, stiffness and weakness in the right thigh was the result of this lesion.

The patient is improving slowly. Surgical intervention was considered but postponed, at least, for the present.

DISCUSSION

Dr. W. B. Cadwalader: A number of years ago, Dr. Spiller showed me a patient almost exactly like Dr. McIver's. The man had been injured with a blackjack in the parietal region, and there was slight weakness on the right side, with pain; and the question came up as to how to explain the pain in the palm of the hand and wrist, and what relation it had to a lesion of the parietal lobe.

In that particular case the pain proved to be purely hysterical in origin.

Dr. Alfred Gordon: There are two diagnoses in my mind in such a case, hysteria and a lesion of the thalamus.

About three months ago, I presented a woman to this society who, following a trauma, developed pain in the right arm, with a paretic condition, but without atrophy. She could not dress herself nor do fine work. The diagnosis was a thalamic lesion of vascular origin, a possibility in Dr. McIver's case.

The other diagnosis in the present case is the one which Dr. Cadwalader mentioned, hysteria.

Dr. McIver, you said that your patient had contraction of visual fields. What kind was it?

Dr. McIver: Concentric.

Dr. Gordon: The fact that this patient is improving and the condition of the visual fields make me lean to a diagnosis of hysteria.

A Case of Optic Atrophy Following Encephalitis. Dr. H. H. Hart.

Among the rarest of the myriad sequelae of epidemic encephalitis is optic neuritis which, while being but an evidence of the ubiquity of the infection is of sufficient rarity to merit some mention. One of the first to report the condition was Smith of Philadelphia, who presented a case to this society in February, 1919. In this case papilledema was found with 5 diopters of swelling in each eye. Holden, an early observer, regarded it as a rare symptom of encephalitis, a concomitant of unusual complications and usually the result of increased intracranial pressure. Spaeth observed five cases of encephalitis with

various ocular manifestations and reported one with binocular optic neuritis of a mild type. Richard reported five cases and Symonds, in the *Lancet* of 1920, reported four cases of bilateral optic neuritis, observed in an American hospital. In 1923, Spiller reported three cases all of which were in boys of about 16 to 17 years of age. In a report of 1,250 cases of encephalitis in England and Wales in the years 1919-1920, the fundi were examined in 234 cases and of these 20 per cent showed eye changes while 5.5 per cent showed optic neuritis. In only two of these cases, however, was the condition called choked disk. In 1922, Libby, after reviewing the literature on the subject, presented a case in which a 5 to 7 diopter swelling was present in a man, aged 22, and the outcome was complete optic atrophy.

The case which we report is that of a girl, aged 10, whose family history is essentially unimportant and whose past history contains the mention of bedwetting until the age of 6 and some jumping out of bed during sleep. She has shown good progress at school, having attained the fourth grade. At the age of 1 year, she had cervical lymphadenitis and at 2 years a tonsillitis with purulent discharge from her right ear which cleared up subsequently. At the age of 8, her tonsils and adenoids were removed.

The present condition dates from about Feb. 28, 1924, when at the age of 9, she was seized with fever and vomiting. She became in three days gradually blind until she was unable to see light. At the same time she complained of a severe headache over the frontal region. The temperature ran from 99 to 102 F., and examination of her eyes at this time showed both pupils dilated and inactive to light. The disks were choked to the extent of 5 diopters. Fine nystagmoid movements were at this time observed by the oculist and her vision as tested by him gave 20/200 in both eyes. For three weeks the fever, eye condition and vomiting remained unchanged, and it was during this period that she showed somnolence and inattention to things about her, and to her parents she seemed to sleep more than usual, especially in the daytime. There were no diplopia, strabismus, ptosis or other ocular manifestations.

After three weeks the temperature and general condition of the patient returned to normal, but she began to vomit frequently for two weeks and then stopped. The swelling of the disks gradually disappeared and the vision improved. Examination at this time showed a vision of 20/20 in each eye. The blood and spinal fluid Wassermann reactions were negative. The leukocyte count was 10,000. Examination of the sinuses was negative.

In August, 1924, vision again gradually failed and her physician, perceiving signs of secondary optic atrophy, sent her to the University of Pennsylvania Hospital, to which she was admitted on August 26. Vision was found to be 20/150 in each eye. There were no nystagmus and no ocular palsies, and the pupils dilated equally and evenly with homatropine hydrobromide. The right disk was decidedly pale with well defined borders. The arteries were perhaps too small as compared with the veins. No distinct changes in the choroid or retina were found. The left eye was similar to the right, the disk was pale but the capillarity was better than that of the right eye. The fields were full but the patient could not recognize green, while red looked to her like yellow. Blue, however, was called properly.

At the hospital neurologic examination showed nothing abnormal save that the left knee jerk was a little more prompt than the right. There was slight albuminuria. On August 28, she was seen by Dr. Spiller, who considered that the patient had had an attack of encephalitis and that the eye condition was a result of it, though in view of the rarity of the condition other causative

factors should be excluded. The nose and throat examination at this time gave negative results. The Wassermann reaction was negative and the roentgenograms of accessory nasal sinuses were also normal.

On February 26 of this year, the patient was sent to the Wills Eye Clinic with the complaint of failing eyesight of one month's duration. She would have to look sideways at objects in order to see them clearly. No loss of color vision was complained of. In walking she would run into objects, saying that she could not see them. At our clinic the left disk was found to be slightly hazy with a slight elevation of the nasal border. The margins of the right disk were found well defined and the capillaries normal. A diagnosis of postneuritic atrophy was made. On the same day she was admitted to the Philadelphia Orthopaedic Hospital and examination showed her to be moderately nourished, about 11 years of age, and with nothing abnormal in the general physical condition. The cranial nerves were normal. There was no impairment of the field of vision. A slight incoordination of the left finger-to-nose test was the only neurologic observation. Examination of the eyes by Dr. Langdon showed a vision of 5/40 in the right eye and 5/5 in the left eye. The pupils were equal and normal. The disks were very pale. The urine and blood examinations gave negative results. A roentgenogram showed the sella turcica to be normal. She was discharged on March 8, with instructions to be brought back in a month's time for further study. Owing to the inability of her father to comply with our request, we have been unfortunately unable to present the girl in person before the society.

I believe that one or two features of the condition are worthy of comment. Holden believed that optic neuritis and papilledema was caused by increased intracranial pressure in encephalitis. In this case it is quite probable that the neuritis was primarily due to this condition for it was observed at the same time that the patient complained of headache and vomiting. However, in some of the cases reported, these symptoms of increased intracranial pressure were absent and it is possible that a meningitic process would be the causal factor.

A feature noted in many of the cases reported has been a fluctuation in the degree of optic neuritis and this has been noted sufficiently in this case in which remissions and exacerbations of the condition have been observed since the onset in February, 1924.

CEREBRAL SYPHILIS OCCURRING SEVEN MONTHS AFTER INITIAL LESION; THERAPEUTIC RESULT AFTER INOCULATION WITH MALARIA. DR. GEORGE WILSON.

A white girl, aged 18, with an unimportant family history, contracted syphilis in June, 1925, and had a secondary eruption in August. She was treated as follows: bichloride of mercury 1/24 grain (0.0027 Gm.) and potassium iodide 5 grains (0.324 Gm.) three times daily; mercurial ointment 60 grains (3.88 Gm.) thrice weekly. Beginning August 15, she was given neo-arsphenamine 45 grains (2.9 Gm.) for seven weeks when her blood was negative and all medicine was stopped. On December 24, the blood was again positive and she was given six more injections of neo-arsphenamine, when the blood was again negative.

In January, 1926, she suffered from severe headaches and had two convulsions. Shortly after this she developed tinnitus in the right ear, and double vision. The headaches were present only at night.

Examination.—The patient was seen on March 23, 1926, when she had a complete right sixth nerve palsy, was partially deaf in the right ear and complained of tinnitus; otherwise the neurologic examination gave negative results. On March 24,

she was examined by Dr. A. G. Fewell, who made a diagnosis of optic neuritis. Dr. Fewell reexamined her on April 21 and said that the eye condition had not changed, and at that time he concluded that the condition was not optic neuritis because there had been no definite change since the last examination; he then thought it was a pseudoneuritis.

The blood Wassermann reaction was 4 plus; the spinal fluid Wassermann reaction was 3 plus, and the colloidal gold curve was as follows: 125555 321000. There were 573 cells to the cubic millimeter of which 97 per cent were lymphocytes, and a faint trace of globulin was present.

She was inoculated with malaria intravenously on March 25, had a rise in temperature the following day, and subsequently had nine chills. The fever was terminated on April 13. After her third or fourth chill the headache disappeared, and the sixth nerve palsy began to clear up. By April 1 the sixth nerve palsy was practically relieved. The blood was positive for malaria on March 29.

Until the present, there has been no return of the symptoms; she has had no treatment from us with the exception of the fever.

Book Reviews

OLD AND NEW VIEWPOINTS IN PSYCHOLOGY. By KNIGHT DUNLAP. Pp. 163. St. Louis: C. V. Mosby Company, 1925.

This volume contains three public lectures, in as many chapters, on subjects of interest to the psychologist and the psychiatrist, presented in very readable style. The first chapter deals with mental measurements; the second is a discussion on present day schools of psychology; the third is a presentation on the subject of psychologic factors in spiritualism. In two remaining chapters are discussed the psychology of the comic, and the reading of character from external signs.

In the paper on mental measurements the author indicates the existing need for mental tests, particularly in their application to the study of individual psychology in contrast with general psychology, and discusses the historical development of tests. The important fact is pointed out that what we want to know about the patient tested is his capacity to learn and to acquire the knowledge and habits demanded of him; this function is supplied by the Binet-Simon tests for the reason that they measure acquisition almost exclusively; from the measure of what the person has actually acquired we estimate his capacity to acquire.

The reader is wisely cautioned that competence in handling the tests is only to be gained after several years of training and supervision, for "Intelligence testing is the work of a specialist and requires a specialist's training"; furthermore, "In spite of all too general incompetence in administration and interpretation, and in spite of the acknowledged fact that the tests are far from perfect, the Binet-Simon tests have proved their worth, and are now essential for the classification of children with respect to intelligence."

With respect to the alpha and beta tests developed and used in the examination of army recruits, Dunlap states, "It is unfortunate that a great deal of misuse has been made of some of the army testing work, having arisen from the misinterpretation of the meaning of the results." The author is skeptical concerning the value of tests devised as a means of predicting collegiate success for the reason that sufficient investigation of the data obtained and of the students themselves is yet to be made.

"Intelligence testing is not the most important part of mental measurement. . . . We must not forget that the important work before us is the measurement of special aptitudes . . . not only for the educational practice . . . but also for the field of commerce, industry, and labor, in a much wider way." The problems of criminality and immigration are still under investigation. The vital need throughout the entire field of mental measurement, including intelligence testing, is the promotion of research, for the reason that of late too much emphasis has been placed on the routine application of the tests, and their merely statistical evaluation.

In chapter 2 the author first distinguishes between individual psychology and general psychology, the former being little dependent on speculation as the data are concrete and require the application of the same scientific methods as are utilized by the chemist and physicist. General psychology requires, on the other hand, working hypotheses. It is to this fact that the difficulties which have arisen are due, partly because there is a tendency to go beyond the requirements of science and "inject" into psychology speculative theory

which is foreign to the subject; in fact, it is easier, says Dunlap, for men in other fields to do this than for psychologists. Because psychology needs the other sciences, deference is paid by psychologists to the metaphysical speculations of leaders in biology, physics and philosophy.

There follows a statement of the situation arising from the struggle of psychology to emerge from the influence of "introspectionalism" and "behaviorism" to which is added a discussion of the methods of the latter school and its point of view.

Along with, or following, these schools which have sprung from the older "idea psychology," has arisen the freudian movement which has gained its strength because it deals with sex, and because it offers a means of cure to be classed with "New Thought," "Christian Science" and other cults. What the older psychologists called the "idea" Freud calls the "wish," and the whole scheme of psychologic explanation of mental phenomena on the basis of "repression," "censorship," and so forth, is intended to be metaphoric. "If this were always the case, there would be no harm done." "There is no objection to the construction of allegories. . . . Whenever allegory is mixed with attempts at science, we become suspicious of the mixture, and our suspicions are usually justified." So far, states Dunlap, the freudian system is a mere clever combination of abandoned parts of old psychologic machinery, and he (Dunlap) regards acceptation of the hypothesis of the "unconscious" as unscientific, and believes that the doctrine belongs strictly to religion and not to science.

Next, the author discusses the speculations which philosophers and biologists have "injected into psychology," with special reference to the conceptions of "instinct" and "intelligence," noting that psychology now uses the term instinct as "an abstraction covering the fact that the various reactions of which an animal is capable have a basis in heredity, and that the animal is therefore, to a certain extent, predisposed to certain lines of action upon certain stimulations; predisposed through the normal growth and development of the organism itself." The term "intelligence" is similarly used as an abstraction.

Recognizing that McDougall has made in total as great a contribution to the science as any living psychologist, and, after entering on an explanation of McDougall's "instinct-psychology," Dunlap states, "The instinct psychology is, in effect, another example of the same manifestation of the same tendency which is exhibited in the old 'faculty psychology,' and in the Freudian system: namely, to abstract from a group of phenomena a characteristic, give it a name, and explain the phenomena by saying they are manifestations of the abstraction named." While the instinct is generally rejected as an explanatory concept, Dunlap regards as unfortunate the fact that "by some extremists it is repudiated even as a useful abstraction." When we wish to emphasize the undoubtedly innate or hereditary basis of reactions and activity, instinct is a useful abstract term. "We do not assume that any action of an animal after emerging from the uterus or hatching from the egg is purely instinctive." "As for the instincts, in the plural . . . . they are at best classifications to suit the purpose of the psychologists. . . . . They may be useful for reference, but no more explain psychological phenomena than a filing system in the office of an insurance company explains the business of insurance."

The author closes the chapter with the statement. "In the great field of general psychology as well as individual psychology we deal with facts, and as the science of psychology becomes more and more distinct and unified, the theoretical problems which the other sciences thrust upon us become less

and less conspicuous." But at present the psychologist needs to be somewhat skilled in speculation in order to avoid it efficiently.

In the chapter on psychologic factors in spiritualism, the author indicates that he is not sure that a great wave of spiritualism has swept the country, as is believed by many interested persons. In fact he is inclined to the belief that "there is less hesitancy over the admission of interest and less fear of the result of the confession of faith." The interest to the psychologist lies in the fact that it offers a chance of adding to our knowledge of human nature —and an opportunity for social service in mental hygiene. The phases of the subject considered as important are: 1. Why people are so eager to accept the claims of spiritualists in spite of the lack of authenticity of its claims; if they were authenticated they would throw little light on the question of immortality. 2. How is it possible that some men of good reputation in science can endorse these claims if they have no scientific backing? 3. Are any of the mediums sincere?

The second question is attacked first, with the conclusion that the scientific men who have openly accepted the claims of spiritualism were in the main physicists who are not qualified psychologically to explain the phenomena believed to be observed, for the reason that the more intensively they specialize in mathematical physics the less likely are they to obtain training in psychology. "In these cases he is at a disadvantage in the handling of data which cannot be mathematically treated. . . . The best combination for investigation in psychical research would be a trained psychologist and a Burns Detective." The importance of "credulous belief," which involves "a powerful drive of an emotional sort," and the fact that the interpretations wished for are put on the communications received, are emphasized as outstanding factors. Dunlap believes that "many mediums are sincere, at least moderately sincere, and that even some of those who deliberately cheat and invent in their messages, really believe that a part of their communications come from a source outside of themselves." The real explanation of their claims seems to be based on something as yet unexplained; namely, in "a device of those who do not understand the mechanism of the mind, and hence cannot find a real explanation."

Under the caption "The Psychology of the Comic," a statement is presented on the subject of the psychology of the origin of laughter which has been considerably discussed in psychologic literature. Unfortunately, it is much easier to indicate the causes of laughter, and to describe biologically the mechanism of the function of laughter than it is to explain the phenomena on a purely psychologic basis. In the comic, Dunlap includes ten different groups of situations in which the dominant note exists in the fact that things, persons and animals are inferior, which becomes manifest to the observer. The first part of the chapter covers the question "as to what we laugh at." The question as to why we laugh is treated much more briefly, and is summed up as "the comic is always that which enhances one's appreciation of his own superiority, but not all suggestions of superiority are comic."

"The Reading of Character from External Signs" refers in a large measure to the efforts which have been made by the employment of nonscientific methods purporting to analyze human character. Dunlap states that the important question is "what are the signs which tell us something about the mental characteristics of other persons?" He concludes that the changes are physiologic rather than anatomic, and that only the "most gross of these signs have been analyzed. . . . . Although we do not know that it is possible to develop a science of character estimation, serious work in the attempt to find out is

highly desirable. . . . . In the meantime, in the interests of the gullible public as well as in the interest of psychology, an educational campaign against 'character analysis' is imperative."

On the whole this volume contains a great deal that is of interest and value to one who is interested in the various controversies in the field of psychology at present, presented by a psychologist who is evidently determined to follow the straight and narrow path required in all inquiry of a scientific nature.

ANATOMIE CÉRÉBRALE. NOYAUX GRIS CENTRAUX ET LA RÉGION MÉSENCÉPHALO-SOUS-OPTIQUE. By C. FOIX and J. NICOLESCO. Price, 125 fr. Pp. 581. Paris: Masson & Cie., 1925.

This is an exceedingly interesting and unusual book. It is most timely in that it discusses the striate body and midbrain which at present are arousing great interest. The book is divided into three parts. In the first there is a general anatomic survey of the caudate nucleus, lenticular nucleus, optic thalamus, the region of the third ventricle, sublenticular and subthalamic regions; the midbrain including the red nucleus, locus niger and the commisures. Then follows an ingenious alphabetic index with definitions of the principle formations of the gray and white structures. Finally the embryology of these structures is discussed.

The second part consists of a photographic representation of serial sections, showing in the first part the fiber formation, while the second demonstrates the cytology, the cuts being made in three directions, vertical, frontal, horizontal and sagittal. Each myelinic cut is represented by a photomicrograph, opposite which is a chart which explains it. Each cut is accompanied by an adequate explanation. The cytologic illustrations, on the other hand, are represented by schematic drawings with accompanying explanatory text. The first and second parts have altogether 180 illustrations.

The third section discusses the cellular structure and myelinic connections with a general summary of the functions of each part. For example, in the discussion of the optic thalamus there is first a general introduction followed by a discussion of the cellular structure, in which the various nuclei are beautifully described. There are two colored plates which show some of the finer cell formation. This is followed by a description of the myelinic structure of the thalamus and its connections with the surrounding parts. Then there follows the embryology, comparative anatomy, myelinization, and finally the vascularization of the thalamus, with a short statement of its function. This plan is followed throughout.

Too much praise cannot be given for the conception of the work. The illustrations are beyond praise. There are six color plates, five of which show the finer cellular structure of the organs discussed, and the sixth plate gives a schematic drawing of the connections of the striate body with the cerebrum and cerebellum. This drawing follows along the lines of Vogt and others. Finally there is a pathologic anatomic study of five cases of Parkinson's disease. This book should be in the hands of every laboratory and every student of neurology.

A STUDY OF AMERICAN INTELLIGENCE. By CARL BRIGHAM, PH.D. Cloth. Pp. 210. Princeton: Princeton University Press, 1923.

In a foreword Dr. Robert M. Yerkes states, "Mr. Brigham has rendered a notable service to psychology, to sociology, and above all to our law-makers

by carefully re-examining and re-presenting with illuminating discussion the data relative to intelligence and nativity first published in the official report of the psychological examining in the United States Army." The greater portion of the book consists of a restatement of the data obtained by tests of the draft army in the World War, together with the statistical treatment of these data. On the basis of his results the author draws comparisons regarding the relative intelligence of the various racial groups represented in the draft. These would appear to corroborate the widespread conviction of the superiority of the Nordic element in our population over the Alpine and Mediterranean elements and over the American negro.

Brigham concludes that American intelligence is declining, and will proceed with an accelerating rate as the racial admixture becomes more extensive, unless public action be aroused to prevent it. Under the head of such preventive action he includes: (1) not only restrictive but highly selective immigration; (2) prevention of the continued propagation of defective strains in the present population.

The tests made in the army during the World War have been the subject of many statistical studies, to which this book is a worthy addition. Dr. Brigham's association with the Surgeon General's office gave him an unusual opportunity for study of the data. With his conclusions, likewise, there can be no quarrel, provided we are careful in our thinking to observe the limitations which the author himself has set. He emphasizes definitely the point that his study applies strictly to those representatives of various European racial groups who have, for one reason or another, chosen to migrate to the United States. It is obvious that no valid deductions can be made regarding the intelligence of any one of these groups as a whole, since we have no reason for assuming that those portions of the race which tend to emigrate are a fair sample of the entire race. On the other hand we are probably safe in expecting that the same selective factors — social, economic, etc. — will continue to be operative, and that there will be little essential change in the intelligence of future immigration groups.

Dr. Brigham has contributed certain useful facts to a problem which has too often been treated with mere speculation and assumption. It is improbable that his suggestions for palliation will be put into practice at any time in the immediate future; it is desirable, however, that the necessity for some such measures be kept in mind.